The Journal of Thoracic Surgery

VOL. 17 APRIL, 1948 NO. 2

Original Communications

SURGICAL MANAGEMENT OF CARCINOMA OF THE LOWER TWO-THIRDS OF THE ESOPHAGUS AND CARDIAC END OF THE STOMACH

JOHN W. STRIEDER, M.D.

BOSTON, MASS.

NOTABLE achievements have been made in the surgical treatment of various lesions of the esophagus during the past eight years. This is particularly true of carcinoma of the esophagus when it occurs at or below the level of the top of the arch of the aorta.

When one considers that up to 1943 the only five-year cure of carcinoma of the esophagus was achieved by Torek[1] as a result of his successful operation in 1913, it is understandable that in 1940 Garlock[2] could write, "Heretofore, the diagnosis of carcinoma of the esophagus was tantamount to signing the patient's death warrant, and the physician advised palliative measures consisting of gastrostomy, radiation, or both." Because of the pioneer work of Adams and Phemister,[3] Garlock,[2] Churchill and Sweet[4] and others, such a situation no longer exists. Now the operative mortality for resections of carcinoma of this portion of the esophagus is not prohibitive, and it is being steadily lowered. The aggregate of figures published even recently cannot reflect the true picture, which is a fluid one and is constantly improving, so that recent operations resulting in probable cures will not be apparent as end results for some years. Moreover, as physicians become increasingly aware of the availability of surgical cure or palliation, more cases with earlier diagnosis will be sent to the thoracic surgeon for treatment, with a consequent improvement of end results.

In my opinion, the technical maneuvers that have given impetus to the increasingly satisfactory surgical treatment of cancer of the esophagus at or below the arch of the aorta, are the demonstration of the remarkable adaptability of the stomach to the thoracic cavity after adequate mobilization, and

From the Thoracic Surgical Services of the Boston City and Massachusetts Memorial Hospitals.

Read at the Twenty-seventh Annual Meeting of The American Association for Thoracic Surgery, St. Louis, Mo., May 28, 29, and 30, 1947.

Garlock's[5] suggestion that the proximal segment of the esophagus be brought over and anterior to the arch of the aorta. By these means, esophagogastric continuity has been rendered feasible in a much larger group of higher lesions, and the known disadvantages of the Torek procedure have been obviated.

Churchill and Sweet[4] have suggested that the esophagus be considered in fourths, rather than the usual upper, middle, and lower thirds, and this serves a useful purpose in evaluating the problem of esophageal cancer from the anatomical aspect. As a matter of immediate practical significance, the location of all esophageal cancers at or below the arch of the aorta is important only as regards the necessity for completing the esophagogastric anastomosis either below the arch, leaving the proximal esophagus in situ, or above the arch, by bringing the proximal esophagus anterior to the arch. Frequently tumors located in the lower portion of the "midthoracic" esophagus may be resected with a safe margin, and esophagogastric anastomosis may be performed below the arch. The postoperative course is no different from tumors of the lower one-fourth similarly treated. It is on this vital point, in my own experience, that the survival rate hinges. Thus the operative mortality is from three to five times greater if the proximal esophagus is brought over the arch than if the same segment can be left safely in situ for anastomosis. It is suggested that future communications include this classification in reporting results.

The purpose of this paper is to report the author's experience with seventy-one patients with carcinoma of the esophagus and cardiac end of the stomach who were operated upon and of whom forty-six patients had forty-seven carcinomas which proved to be resectable, one patient being operated upon twice (case report). No claim for originality is made in any of the procedures described. The published works of the select group of pioneers in the subject have been drawn upon freely.

Although the operative results of the entire series is reported, it seemed desirable for purposes of contrast to break the series down into two groups, those prior to 1945 and those from 1945 to May 1, 1947. By way of partial extenuation of the early poor results, it may be said that many factors in the postoperative care during the war years were incredibly bad and during 1944 the problem was largely abandoned.

SELECTION OF CASES

Age.—The youngest patient who had successful resection was 52 years old; the oldest was 84 (Fig. 16). In no instance was a patient denied operation solely on the basis of age. It was our feeling that the general condition of the patient, and, in particular, that of the cardiovascular apparatus was of more importance than the chronological age. In one instance, a 72-year-old patient was operated upon while fibrillating. Fibrillation persisted during the entire operative and postoperative periods and the patient left the hospital in good condition.

Lymph Nodes.—As has been pointed out,[4] the involvement of cervical lymph nodes is relatively infrequent except in lesions high in the esophagus. In lesions below the arch, the lymphatic drainage tends to the periesophageal

nodes, the subphrenic nodes, and the nodes in the region of the coeliac axis and left gastric arteries.

Metastases.—Metastatic lesions in the lungs should be carefully searched for by roentgenologic examination of the chest. In low esophageal cancers and cancers of the cardiac end of the stomach, rectal examination may reveal metastatic deposits in the pelvis. Involvement of the liver in esophageal lesions is a relatively late manifestation. When we have encountered it in high gastric lesions with involvement of the esophagus, we have proceeded to resection not withstanding, for reasons of palliation. The tendency for the primary esophageal lesion to spread in the wall of the esophagus is well recognized, so that a relatively wide excision of the tumor should be made when possible.

Esophagoscopy.—We have operated upon only one patient with cancer of the esophagus without a positive tissue biopsy obtained at the time of esophagoscopy. When the esophagus is involved by high gastric cancer, it is frequently possible to make the diagnosis by biopsy through the esophagoscope. However, in this latter group, if a positive tissue diagnosis cannot be made, exploration is undertaken on the basis of roentgenologic findings or on suspicion alone.

Bronchoscopy.—At the time of esophagoscopy, a routine bronchoscopy is also performed. On at least four occasions, we have found the tumor invading the left stem bronchus or the trachea, which has obviated the necessity for exploration.

Roentgenological Examination.—Even when the esophageal cancer appeared extensive by this method of examination, in most instances we have chosen to explore the patient in order to give him the benefit of the doubt, although frequently we have only confirmed the x-ray findings.

PREOPERATIVE PREPARATION

Every effort is made to improve the patient's general condition, and to bring him to operation under optimal circumstances for his case. The oral hygiene is investigated by the dental consultant and careful prophylaxis and scaling is done. If any of the teeth are so badly involved as to preclude safe anesthesia, they are extracted. The blood picture is evaluated, and transfusions are given as necessary. Adequate parenteral fluids with amino acids are given in order to bring the blood protein up to a safe level. By the careful parenteral administration of fluids and electrolytes as well as the protein components, patients can actually be made to gain weight. Forty-eight hours before operation the administration of penicillin parenterally in doses of 50,000 units every three hours is begun. If there is complete or almost complete obstruction of the esophagus, lavage of the esophagus with half-strength Dakin's solution is undertaken twice a day beginning three or four days before the proposed time of operation. In a few instances, we have performed preliminary jejunostomy in order to improve the patient's nutrition preoperatively. Immediately before operation a continuous intravenous infusion is started, and in most of the cases the patients received blood continuously during the operation by one or

more veins, as seemed indicated. Patients frequently receive from 1,000 to 2,500 c.c. of blood during the course of the operation.

ANESTHESIA

Anesthesia is induced with intravenous Pentothal and nitrous oxide and oxygen. Curare is given to facilitate the introduction of the intratracheal tube. The patient is then carried on intravenous pentothal with supplementary nitrous oxide and oxygen until the pleural cavity is opened, when the change is made to ether vapor and oxygen, on which he is maintained for the remainder of the operation. The use of the nonexplosive anesthetic in the early part of the operation permits the use of the endothermic coagulating current to secure hemostasis of the skin, subcutaneous tissues, and extracostal muscles. During the course of the operation, the lung is expanded at fifteen to twenty-minute intervals in order to facilitate re-expansion at the end of the operation.

OPERATIVE TECHNIQUE

A. *Carcinoma of the cardiac end of the stomach and carcinoma of the esophagus so situated that esophagogastric anastomosis may be made below the arch.*—The patient is placed in the right lateral decubitus position. A curving incision is made over the left ninth rib from the costochondral junction to within a centimeter or two of the spinous processes. If the carcinoma is situated in the esophagus and somewhat above the arbitrary division of the lower fourth, it may be better to select the eighth rib. In either case, the rib just above and below the rib selected for resection is divided by resecting subperiosteally a one-centimeter length just lateral to its respective transverse process. The intercostal nerve of the rib selected for resection is injected with one per cent Nupercaine in oil, as are two or three intercostal nerves above and below this rib. The rib selected is resected from the transverse process forward to within a centimeter or two of the costochondral junction. The pleural cavity is entered through the periosteal bed of this rib. Pads moistened with warm saline are placed over the wound edges and the Finochietto rib spreader is placed. Excellent exposure is thus obtained (Fig. 1). In none of our cases have we found it necessary to extend the incision across the chondral arch, so as to make a combined thoracoabdominal approach. The pulmonary ligament is now divided up to the inferior pulmonary vein and any bleeding vessels are secured. If the tumor is in the esophagus proper, the mediastinal pleura is incised, and, if possible, the esophagus is freed just above the hiatus and a tape passed around it. Before opening the diaphragm, operability of the tumor in the esophagus is determined, and by careful sharp and blunt dissection, the tumor is freed from the surrounding structures and dissection is carried upward around the esophagus for a suitable distance so that anastomosis may be made. There is relatively little blood supply to this segment of the esophagus, so that very few branches of the aorta are encountered, but when they are encountered they must be divided and ligated. No attempt is made to preserve the vagus nerves, which are sacrificed as necessary. If the tumor is operable, the phrenic nerve is now crushed as it lies on the pericardium just above the diaphragm. The diaphragm is opened by an incision extending from its lateral portion into the

esophageal hiatus, during the course of which the subphrenic artery is secured and suture ligated (Fig. 2). If the primary tumor is in the stomach, the diaphragm may be opened at once with little preliminary dissection of the esophagus above the diaphragm. The vascular connections of the stomach now receive the attention of the operator. The gastrosplenic ligament is divided, and the vasa brevia are ligated. In most instances, it is not necessary to perform a splenectomy; however, if it interferes with mobilization of the stomach, or if there is involvement in the tumor mass, splenectomy is performed. The left gastroepiploic artery is divided, but care is taken to preserve the length of the vessel to serve as an anastomotic channel for bringing blood to the cardiac end of the stomach by dividing the branches in the gastro-colic omentum distal

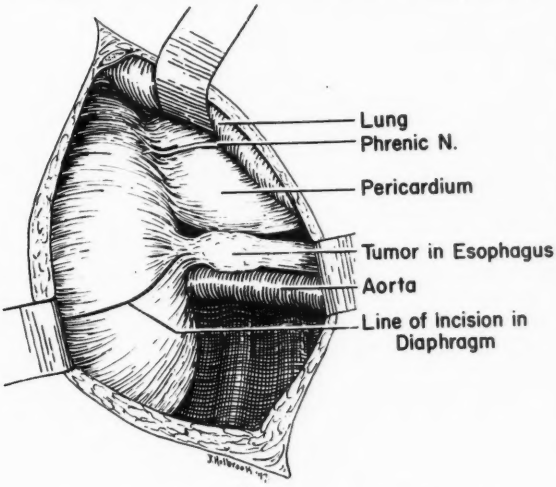


Fig. 1.

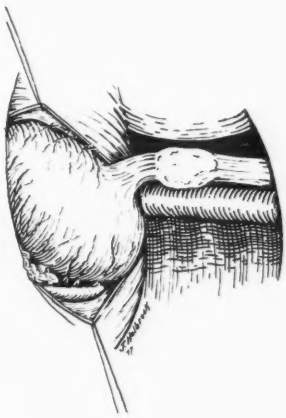


Fig. 2.

Fig. 1.—The left pleural cavity has been entered through the bed of the ninth rib. The mediastinal pleura has been incised and the tumor in the lower esophagus exposed.

Fig. 2.—The diaphragm has been opened from its lateral portion into the esophageal hiatus and the margins retracted by guy sutures. The vascular connections of the upper portion of the stomach have been ligated and divided.

to it. Attention is now turned to the gastrohepatic ligament, which is divided between clamps, and the vessels are secured. In most instances, it is necessary to divide the left gastric vessels, following which the fundus of the stomach can be brought high into the chest. If the tumor is in the stomach, the tumor-bearing portion is resected between clamps and the distal portion is closed by a continuous chromic catgut suture and inverted with a second continuous chromic catgut suture. The suture line is further reinforced by a layer of interrupted silk sutures. If the tumor is in the esophagus and there is sufficient room between it and the cardioesophageal junction, the esophagus is divided between two ligature of No. 2 chromic catgut and the distal end is inverted into the stomach by means of a silk purse-string suture; this is reinforced with a layer of interrupted silk sutures (Fig. 3). The proximal end of the esophagus or stomach is now protected by a piece of rubber dam or a rubber

glove. During the course of the dissection up to this point, all nodes that are encountered in the periesophageal and subphrenic regions and in the region of the left gastric artery are resected in so far as it is possible.

It is important not to resect the tumor-bearing portion of the specimen at this stage of the operation, since the esophagus retracts markedly and anastomosis is thereby rendered exceedingly difficult. Using the clamp on the proximal portion of the stomach or esophagus for gentle traction, the specimen is now lifted up out of the pleural cavity and on to the superior margin of the incision so as to present the mesial aspect of the esophagus for the first layer of the anastomosis with the stomach. The stomach is brought up into the chest, and a circular incision is made through the serosa and muscularis, as shown in Fig. 4.

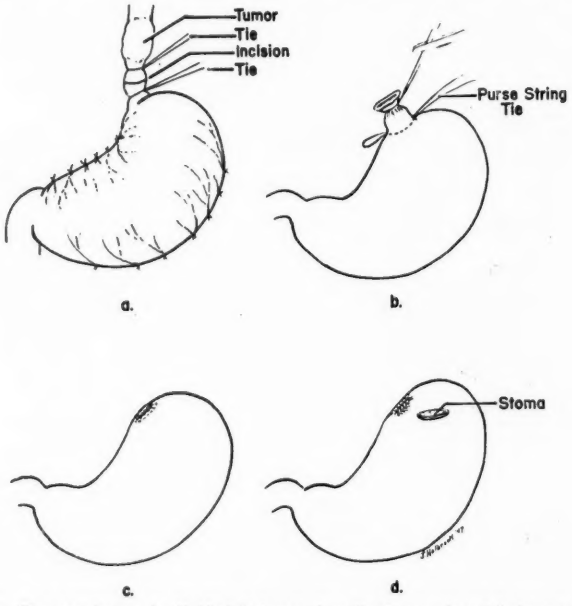


Fig. 3.—*a*, The esophagus is divided between two ligatures placed between the tumor and the stomach. *b*, The distal stump of the esophagus is invaginated into the stomach by purse-string suture. *c*, A reinforcing layer of interrupted silk is placed over the purse-string. *d*, An elliptical button of serosa and muscularis is removed at the site of the new esophagogastric anastomosis.

The button so outlined and composed of serosa and muscularis is dissected free without opening the gastric mucosa and the small vessels are secured by suture ligatures of fine silk close to the margins of the incision. A suitable location on the esophagus a sufficient distance above the tumor-bearing area to be judged above the farthest extent of the cancer is now selected, and the first or posterior row of sutures is placed. A three-layer anastomosis after the manner of Churchill and Sweet[4] using fine silk interrupted sutures with meticulous approximation of the mucosal layers is now made, as shown in Fig. 5. The esophagus is incised layer by layer as the anastomosis is made so that the second layer is muscularis of esophagus to muscularis and serosa of stomach. The esophagus is

then completely divided somewhat on the bias to obtain a larger lumen, and the mucosal layer is completed. Following this, the anterior layer of muscularis of esophagus to muscularis and serosa of stomach is completed and the outermost layer of muscularis of esophagus to serosa of stomach is also completed.

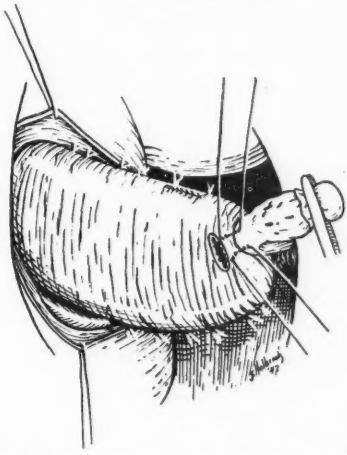


Fig. 4.—The stomach has been brought to the necessary height in the pleural cavity. The protected tumor-bearing portion of the proximal esophagus is brought out of the chest and over the superior wound margin and the first row (posterior) of interrupted silk, approximating muscularis of esophagus to serosa of stomach, is placed.

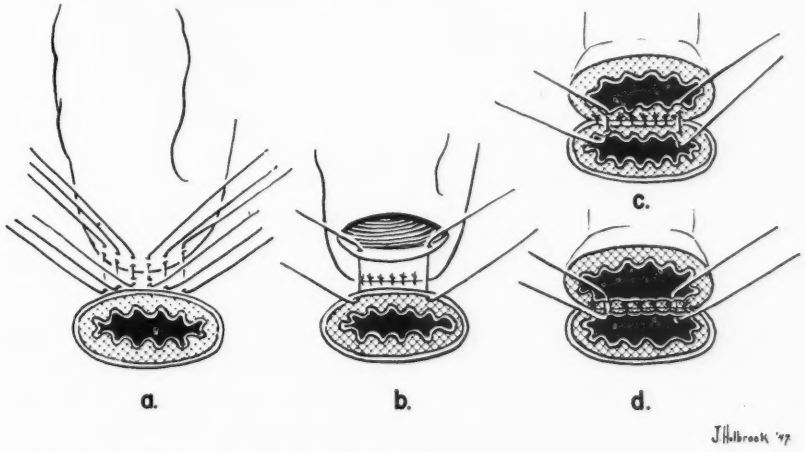


Fig. 5.—*a*, Posterior row of interrupted silk, approximating esophageal muscularis to gastric serosa, is placed. *b*, The second row of interrupted silk approximating cut edge of muscularis of esophagus to cut edge of gastric muscularis and serosa is begun. *c* and *d*, The meticulous approximation of mucosal layers, using fine interrupted silk with knots tied in the lumen, is begun posteriorly and will be completed.

A portion of omentum is next brought up and fixed about the anastomosis. The stomach is sutured to the contiguous pleura laterally by several interrupted silk sutures so as to relieve any strain on the anastomosis (Fig. 6). The diaphragm

is next closed about the stomach with interrupted sutures and the lateral portion of the incision of the diaphragm is also approximated with interrupted silk (Fig. 7). The pleural cavity is flushed out thoroughly with warm normal saline and a fenestrated rectal tube for under-water drainage is placed in the pleural cavity through a lower interspace. The tip of this catheter is held in proper position by a loop of chromic catgut so that when the catheter is removed it will slip freely from the pleural cavity. The chest is now closed in layers after instilling 100,000 units of penicillin, and re-expanding the lung. Closure is effected by using interrupted No. 0 chromic catgut to the pleura and intercostal muscles, interrupted chromic No. 0 catgut to the extracostal muscles, subcutaneous interrupted No. 000 plain catgut and interrupted silk to the skin. After the dressing has been applied, the patient is placed on his back and bronchoscopy is performed so that the tracheobronchial tree is dry when the patient leaves the operating room.

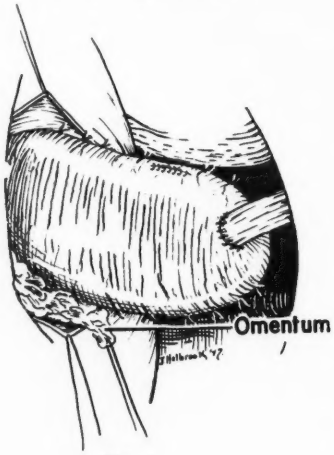


Fig. 6.

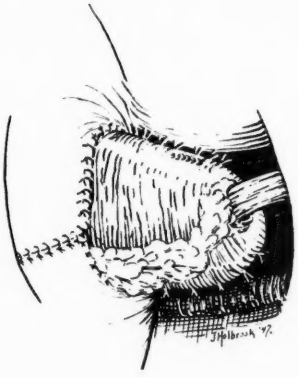


Fig. 7.

Fig. 6.—Anastomosis is completed. Stomach is anchored to contiguous pleura by interrupted silk sutures. Omentum is being brought up.

Fig. 7.—Diaphragm closed around the stomach by interrupted silk sutures fixing the stomach in position and closing possible sites of herniation. Omentum is sutured about anastomosis.

B. *Carcinoma of the esophagus located at or below the arch of the aorta requiring supra-aortic anastomosis.*—When the tumor in the esophagus is so high that it is judged it will be necessary to free the esophagus from under the arch of the aorta and bring it anterior to the aorta for anastomosis, the eighth rib is selected for resection and the seventh, sixth, and occasionally the fifth ribs are divided just lateral to their respective transverse processes in order to secure adequate exposure high in the thorax. The mediastinal pleura is incised and the free portion of the esophagus is delivered into the wound and a tape placed about it for traction. By careful blunt and sharp dissection, the esophagus with its tumor-bearing portion is freed from the surrounding structures. These include the inferior pulmonary vein, the left bronchus, and, on

occasion, the posterolateral aspect of the pericardium in relation to the left auricle. It is frequently necessary to sacrifice a portion of the right pleura, in which case the anesthetist initiates positive pressure and a pack is placed against the defect until the operation can be completed. Dissection of the

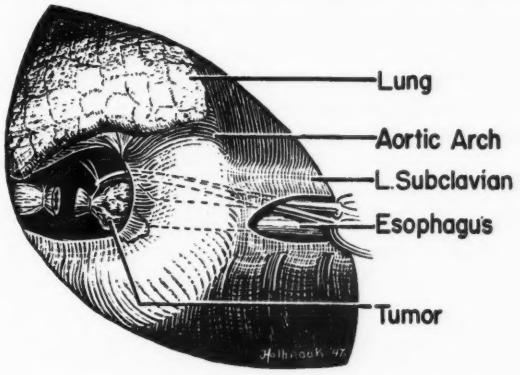


Fig. 8.—The tumor and proximal esophagus have been freed from beneath the arch and the esophagus divided between ligatures. The esophagus is divided lower than shown here so that the distal stump may be inverted into the stomach (Fig. 3). A clamp is indicated being passed deep to the aorta to grasp the proximal ligature and deliver the tumor-bearing portion over and anterior to the arch.

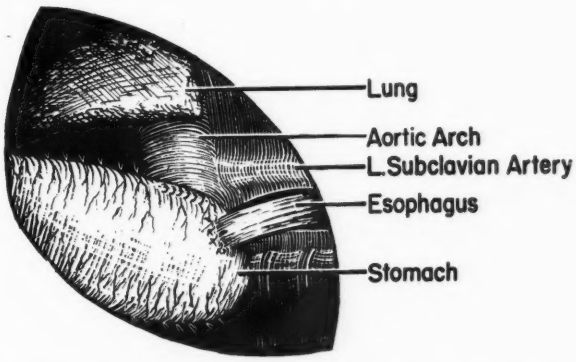


Fig. 9.—Esophagogastric anastomosis completed above and anterior to the arch of the aorta.

tumor and freeing of the esophagus from the under surface of the aortic arch is the most difficult part of this procedure and can be facilitated by working from below and above the arch in turn or simultaneously. It may be necessary to divide a few of the high intercostal arteries in order to turn the arch of the aorta outward and facilitate dissection beneath it. An incision of the mediastinal pleura posterior to the left subclavian artery with delivery of normal esophagus at this level so that a tape can be placed around it frequently aids the dissection (Fig. 8). It is at this level that the thoracic duct is encountered and may be injured. If it is injured and recognized, it should be ligated. If the tumor has

involved the prevertebral fascia, the plane of dissection may lie in the prevertebral space where troublesome bleeding from injury to the anterior external vertebral venous plexus may be encountered. When the esophagus has been freed and the tumor is found to be resectable, the diaphragm is opened, the stomach mobilized, and the esophagogastric anastomosis made as described above (operative technique, *A*). Anastomosis between stomach and esophagus above the arch is frequently difficult because of its relative inaccessibility; as has been pointed out by various authors, it should be made as high as possible in order to take advantage of the blood supply from the inferior thyroid artery, which is the sole blood supply of this portion of the esophagus (Fig. 9).

COMMENTS ON OPERATIVE TECHNIQUE

Incision.—In no instance has it been necessary to extend the incision across the chondral arch or otherwise to make a thoracoabdominal incision of the parietes. Adequate exposure has always been obtained as outlined above.

Avoidance of Trauma.—It is highly desirable, as Sweet[6] has pointed out, that the utmost care be observed in preserving the gastric blood supply. This is accomplished by avoiding the use of clamps or forceps on the stomach and by insuring that the important vessels concerned in the circulation of the stomach are not impaired in the course of division of their vascular connections in the gastrolienal, gastrocolic and gastrohepatic ligaments.

It is also of importance that the esophagus be handled with great gentleness during dissection and when the anastomosis is made. To that end, no clamps or forceps are applied to that portion of the esophagus which will be left behind after anastomosis.

Inoperability as Determined at Operation.—In most instances a condition was deemed inoperable because of invasion of vital structures which could not be sacrificed with the tumor. Inability to separate the tumor from the aorta, bronchus, or inferior pulmonary vein obviously has led to the abandonment of the attempt at resection. No case has been encountered where resection of an invaded portion of the lung would render it operable, although this should be the simplest of procedures.

The spleen and tail of the pancreas have been repeatedly sacrificed and in one case most of the left lobe of the liver has been removed during the resection of a gastric cancer with survival of the patient in apparent good health fourteen months later.

Palliative Resections.—Although it has been apparent at operation that the cancer has spread beyond its original confines as manifested by lymph node involvement or liver metastases, if the primary growth was resectable it has been removed. It is felt that the palliation conferred by restoration of esophagogastric continuity and the ability to swallow food normally during even a short remaining life is ample justification for such an attitude.

Drainage.—In the earlier series the pleural cavity was not drained, reliance being placed on the sulphonamides and penicillin and repeated postoperative thoracenteses. As a result, there was a high incidence of pleural

infections, some of which were fatal. Since routine drainage has been employed, serious pleural infections, except when there has been leakage at the anastomosis, have been negligible.

Bronchoscopy.—The role of bronchoscopy in the preoperative and postoperative care of patients who have been operated upon for carcinoma of the esophagus deserves a special comment. As has been mentioned, it is of the utmost importance that at the time of the esophagoscopy, bronchoscopy also be performed in order to rule out extension of the tumor to the tracheobronchial tree, and, in particular, to the left stem bronchus. At the conclusion of the operation, and after the patient has been turned upon his back, bronchoscopy is again performed so that the patient leaves the operating table with the tracheobronchial tree dry. As in any major thoracic procedure, the patient's inability to raise secretions postoperatively has been a frequent and troublesome complication. This can frequently be aided by intratracheal catheter suction, but in many instances it is necessary to perform bronchoscopy one or more times in the immediate postoperative days.

Complete occlusion of the left stem bronchus with complete atelectasis of the left lung has been encountered in four patients who have had the esophagus dissected from the left bronchus and the anastomosis made anterior to the arch of the aorta. This complication has been discussed by Strieder.[7] In one instance, the occlusion was due to an abscess which had formed in contact with the left stem bronchus as a result of a leak of the suture line. Two other patients died as a result of this complication. In the fourth patient, the edema subsided and the lung subsequently aerated. It has, therefore, been a source of some comfort to us to know that there was not actual involvement of the bronchus by tumor since it had been visualized at least twice before the complication arose.

COMPLICATIONS

Leakage of Anastomosis.—The complication of leak at the site of the esophagogastric anastomosis was the most frequent cause of death in our early series of cases. In the later series, from 1945 to 1947, it has been a rather infrequent complication occurring only three times in the last thirty-four cases. Such leaks occur on approximately the third or fourth day although they have been noted on the eighth day. The complication is usually obvious at once because the patient has marked elevation of temperature and, if the drainage tube is still in place, a foul discharge or particles of recently ingested food are apparent.

A roentgenogram of the chest reveals collapse of the lung because of the leakage of air into the pleural cavity through the anastomosis. Three of the patients who had anterior anastomoses and who developed leaks relatively late recovered. One sealed off the leak immediately and restored esophagogastric continuity and had no further difficulty in swallowing up to the time of his death eight months later from recurrence of the gastric carcinoma. The second was the re-operated case reported in this article. The esophagogastric continuity was restored by secondary healing but there has been troublesome narrowing at the site of anastomosis requiring dilatation at approximately two-month

intervals. This patient, however, is alive and well and eating a full diet one year later (case report). The third patient, a 79-year-old man, developed a tiny leak which apparently did not result in an empyema but was demonstrated by a routine postoperative barium swallow which showed a small extravasation of the barium into the surrounding tissue. The patient is entirely symptom-free and has remained so eight months after operation. The fistulous tract is no longer demonstrable.

Atelectasis.—This has already been discussed, to some extent, under bronchoscopy. All patients exhibiting this complication postoperatively should be treated by bronchoscopy if adequate intratracheal catheter suction does not suffice to permit the lobe or lung to aerate. Where the esophagus has been brought anterior to the arch, the possibility of edema of the stem bronchus resulting from operative trauma must be borne in mind.

Late Strictures.—Narrowing at the site of the esophagogastric anastomosis, without evidence of recurrence of tumor, has been present in four patients of our later series.

Although the complication is an annoying and troublesome one, it has yielded satisfactorily to frequent dilatations and the patients have been able to carry on on a reasonably full diet. The possibility of the stricture being due to recurrence should be considered when the true situation may be revealed by esophagoscopy.

CASE REPORT

The following case is reported in some detail because of the unusual features and because, in so far as can be determined from the literature, it is the first time a patient has had two successful transthoracic partial esophagectomies.

J. N., a 63-year-old man, city park custodian, was admitted to the Massachusetts Memorial Hospitals on May 17, 1946, complaining of increasing dysphagia and loss of twenty pounds in weight of two months' duration. The dysphagia had progressed so that on admission he was able to swallow only liquids and these with considerable difficulty. His past history had no bearing on his present illness; he had always been well and strong. Up to the time of his weight loss, he had been considerably overweight. With the exception of Dupuytren's contracture of both hands, the physical examination and laboratory studies were within normal limits for a man of his age. Roentgenographic and fluoroscopic studies of the esophagus revealed narrowing and ulceration extending from 7 to 14 cm. above the diaphragm (Fig. 10).

On May 18, 1946, bronchoscopy revealed no abnormalities in trachea or major bronchi. At the same sitting, esophagoscopy demonstrated an annular constriction with ulceration 31 cm. from the upper incisors. Biopsy made at this time showed only chronic inflammation of the esophageal mucosa. In spite of the failure to obtain a positive tissue diagnosis, and in view of the typical appearance by esophagoscopy and roentgenography together with the classical history, operation was offered to and accepted by the patient.

On May 23, 1946, left transpleural esophagectomy with esophagogastrostomy below the arch of the aorta was performed without particular incident. It was necessary to free the tumor from the posteromedial aspect of the descending aorta to which it was adherent for a distance of several centimeters. There was no evidence of metastases below or above the diaphragm.

The specimen showed epidermoid carcinoma of the esophagus with tumor cells in the periesophageal fibrous tissue. It appeared, microscopically, that the upper aspect of the tumor had been cut through at operation. The accompanying lymph nodes (subphrenic and periesophageal) were negative.

The patient had a benign postoperative course. He was allowed out of bed on the third and discharged home on the fifteenth postoperative day. On discharge from the hospital, he was eating a full diet with no dysphagia.

He was readmitted to the hospital on June 25, 1946, thirty-three days after operation, complaining of inability to swallow of three days' duration. He had had no difficulty until one week before when rapidly progressive dysphagia had supervened. Esophagoscopy was

Fig. 10.—Roentgenogram after barium meal showing evidence of esophageal carcinoma extending from 7 to 14 cm. above the diaphragm. (Case report.)

performed on June 27, 1946, and again on July 1, 1946. There was out-cropping of tissue at the site of anastomosis which bled easily and the lumen was occluded. Two biopsies showed only granulation tissue. Cautious attempts at endoscopic bougienage were unsuccessful.

Because of the pathologic findings in the specimen removed at the time of resection, the lack of evidence of extension of the growth other than locally, and the excellent condition of the patient at this time, it was decided to attempt further resection.

On July 3, 1946, the left pleural cavity was reentered through the bed of the seventh rib and with considerable difficulty, because of adhesions and indurated tissues, and the original site of anastomosis was resected with a wide margin of stomach and esophagus.

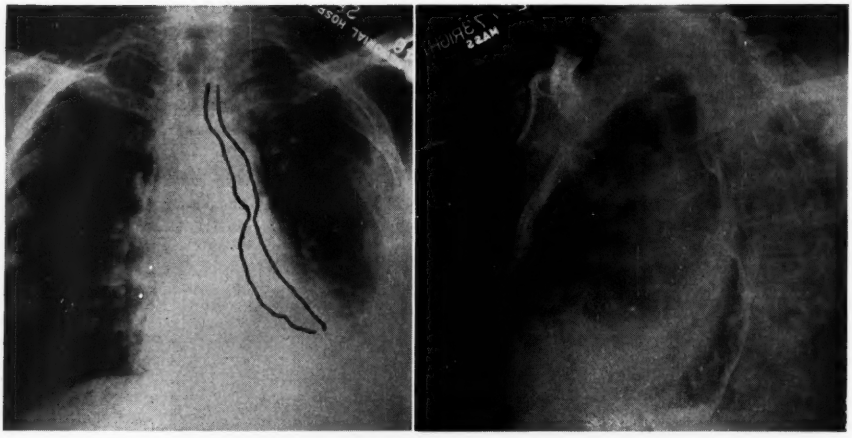


Fig. 11.—Roentgenograms after barium swallow showing restoration of esophagogastric continuity following second partial esophagectomy, secondary healing of partial disruption of suture line, and unroofing of complicating localized empyema.

Fig. 12.—J. N. (case report) photograph taken April 28, 1947, at the time of the last esophageal dilatation. Shown are the scars of the jejunostomy and esophagectomy incisions as well as the healed empyema wound through which the esophagogastric fistula drained.

The proximal esophagus was brought over and anterior to the arch of the aorta and esophagogastric continuity again restored in the new position. There was, undoubtedly, some tension on the suture line because the stomach, after a tedious dissection, could not be mobilized completely satisfactorily.

Careful serial sections of the specimen removed showed no evidence of malignancy on histologic examination.

Three days postoperatively a leak developed at the site of anastomosis. This was confirmed by the appearance of methylene blue in the drainage tube immediately after it was swallowed by the patient. There was associated localized pyopneumothorax, which was

small because of the diffuse pleural symphysis, the result of the original operation, which had been largely undisturbed, purposefully at the time of the second resection. The patient had little systemic reaction and the drainage tube was kept in situ. For the ensuing ten days, the patient's nutrition was maintained by parenteral fluids and transfusions with no essential deviations from normal blood chemistry. However, when it became apparent that there was a reasonable chance for survival, a jejunostomy was performed on July 16, 1946. The patient was then fed a mixture of "Gerolac" (Borden Co.) until he could again swallow with a gratifying maintenance of weight over a period of three months.

The small empyema cavity was unroofed by the resection of segments of several ribs on July 25, 1946. At this operation, the opening at the esophagogastric junction was visualized. There was separation of the anastomosis for about 50 per cent of the circumference. The empyema cavity was revised on Aug. 15, 1946, so as to remove overhanging edges and from this time, there was rapid closure of the cavity and of the esophageal fistula.

During September, 1946, the esophagus was dilated four times under general anesthesia and, with the closure of the fistula, he was able to swallow a full diet at the time of discharge from the hospital, Oct. 5, 1946 (Fig. 11).

Since discharge from the hospital, he has remained well and for the most part has been able to take a full diet except for several days prior to dilatation which has been necessary at intervals of approximately two months. At these times, esophagoscopy has been performed and there has been no evidence of local recurrence. There has been a gain in weight of 30 pounds (Fig. 12). The last dilatation was performed on April 28, 1947. (The patient remains well with no evidence of recurrence in January, 1948.)

RESULTS

An analysis of the total operative experience from 1941 to May 1, 1947 (Table I) reveals that there were seventy-one patients upon whom seventy-two operations were performed. Of these, twenty-five had exploration only, the condition being found inoperable. Forty-seven resections of either stomach or a portion of esophagus or both were performed on forty-six patients. One patient, discussed in brief above, had two resections of portions of the esophagus.

Anastomosis Below the Arch.—There were thirty-five resections of the cardiac end of the stomach or of the lower esophagus or both with esophagogastric anastomosis performed below the arch of the aorta. There were ten hospital deaths in this series, an operative mortality of 24.0 per cent.

TABLE I. TOTAL OPERATIVE EXPERIENCE (1941-1947)

	NUMBER	DIED IN HOSPITAL	OPERATIVE MORTALITY (%)
Total cases	71		
Total operations	72	19	26.3
Exploration only	25	1	4.10
Resections	47	18	38.2
Stomach and low esophagus with anastomosis below arch	35	10	24.0
Esophagus with anastomosis above arch	12	7	63.6
Torek	1	1	100.0

TABLE II. OPERATIVE EXPERIENCE (1941-1944)

	NUMBER	DIED IN HOSPITAL	OPERATIVE MORTALITY (%)
Stomach and low esophagus with anastomosis below arch	10	7	70.0
Esophagus with anastomosis, above arch	2	2	100.0
Torek	1	1	100.0

TABLE III. OPERATIVE EXPERIENCE (1945-1947)

	NUMBER	DIED IN HOSPITAL	OPERATIVE MORTALITY (%)
Stomach and low esophagus with anastomosis below arch	25	3	12.0
Esophagus with anastomosis above arch	9	5	55.5

Anastomosis Above the Arch.—There were eleven resections of carcinoma of the esophagus located sufficiently high in the midthoracic portion so that esophagogastric anastomosis was performed above the arch of the aorta after the suggestion of Garlock. Seven of these patients died in hospital, an operative mortality of 63.6 per cent.

Torek Operation.—There was one patient who had the Torek operation and who died in the hospital.

For purposes of contrast, it is of interest to divide the total operative experience into two series. The first, covering the period 1941-1944 and consisting of only thirteen cases, was accumulated during a period of meager experience and before standardization of procedure and postoperative care. The pleural cavity was not drained and penicillin was not always available. The results were bad. The second, covering the period 1945 to 1947 (May 1), consists of thirty-four cases treated by the two standard operations described above and having the benefit of previous experience in postoperative care. The pleural cavity was always drained and all patients had the benefit of penicillin. The results were considerably improved.

Operative Experience 1941-1944.—There were thirteen operations (Table II).

Anastomosis below the arch: There were ten resections of the cardiac end of the stomach or of the lower esophagus or both with esophagogastric anastomosis performed below the arch of the aorta. There were seven deaths in hospital, an operative mortality of 70 per cent.

Anastomosis above the arch: There were two resections of esophageal cancers situated so high in the midthoracic esophagus that esophagogastric anastomosis was performed above the arch of the aorta. Both of these patients died in hospital.

Torek operation: One patient had the Torek operation and died in hospital.

Operative Experience 1945 to May 1, 1947: There were thirty-four operations (Table III).

Anastomosis below the arch: There were twenty-five resections of the cardiac end of the stomach or of the lower esophagus or both with esophagogastric anastomosis performed below the arch of the aorta. There were three deaths in hospital, an operative mortality of 12.0 per cent.

Anastomosis above the arch: There were nine resections of esophageal cancers situated so high in the midthoracic esophagus that esophagogastric anastomosis was performed above the arch of the aorta. There were five deaths in hospital, an operative mortality of 55.5 per cent.

Survivors: Of the twenty-six operative survivals, twenty-one patients are still alive from one month to fourteen months later, on May 1, 1947, when

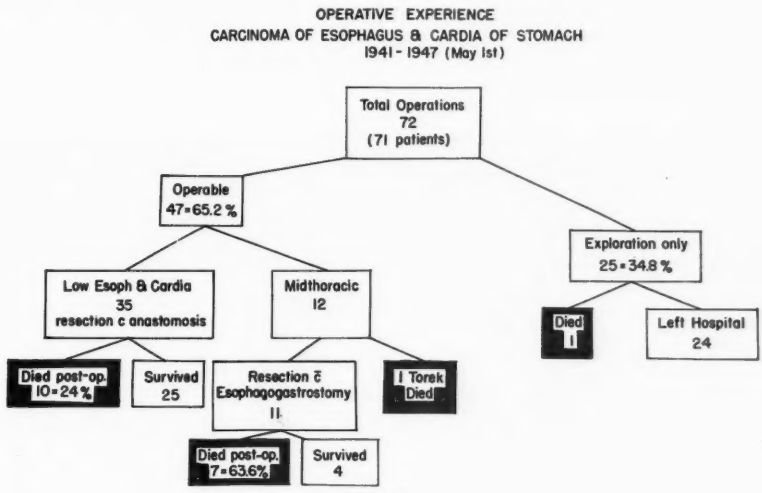


Fig. 13.

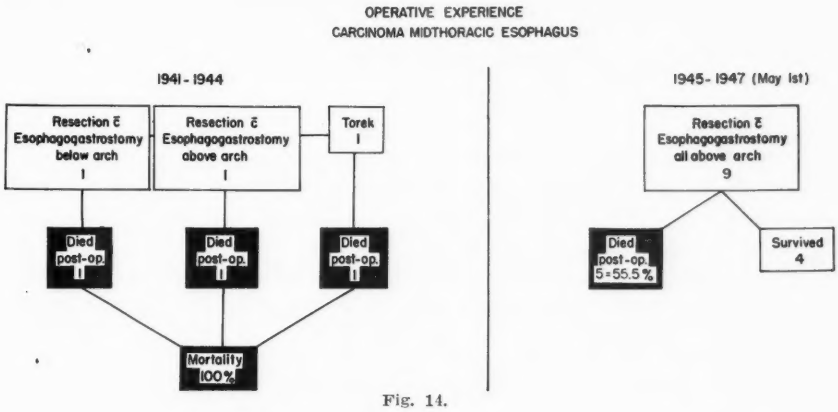


Fig. 14.

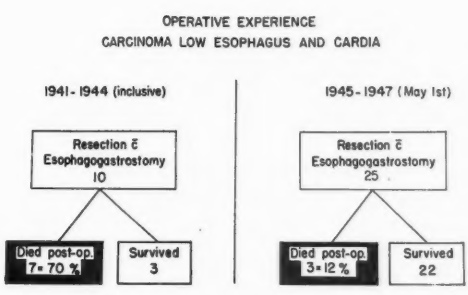


Fig. 15.

this is written (Table IV). One is alive with disease four months postoperatively, after a subtotal gastric resection. The remainder show no evidence of disease clinically, although a few are known to have disease left behind at operation, and others will undoubtedly manifest recurrences sooner or later.

Fig. 16.—I. B., 84-year-old man who had successful transpleural esophagectomy and esophagogastric anastomosis below the arch for carcinoma occurring in the lower midthoracic esophagus. Photograph made six weeks after operation which was performed April 1, 1947. The scars of the operative incision and drainage site are shown in the lateral and posterior views. This patient had Hodgkin's disease of the nasopharynx at age 72, treated by radiation with apparent recovery.

The age distribution of the survivors is shown in Table V. The two oldest survivors are aged 80 and 84 years, respectively. The 84-year-old survivor had histologically proved Hodgkins' disease of the nasopharynx at age 72 treated by radiation and is without evidence of recurrence at this time (Fig. 16). Two other patients in the total series operated successfully had a second primary cancer. One, aged 60 years had a grade III carcinoma of the lip excised seven and one-half months before esophagectomy with anastomosis above the arch, and is now well thirteen months later. The other died one month after transthoracic gastrectomy with a primary carcinoma of the colon.

TABLE IV. DURATION OF SURVIVAL

MONTHS	NUMBER
1-6	9
6-12	8
12-14	4
Total	21

TABLE V. AGES OF SURVIVORS

AGE (YR.)	NUMBER
50-59	5
60-69	9
70-79	5
80-84	2
Total	21

SUMMARY

1. The experience with seventy-two operations on seventy-one patients with carcinoma of the esophagus occurring at or below the arch of the aorta, and of the cardiac end of the stomach, is presented.

2. The preoperative preparation, the operative technique, and the postoperative complications are discussed.

3. It is suggested that in reporting results, the division of cases into (1) those in which esophagogastric anastomosis was made below the arch of the aorta and (2) those in which it was made above and in front of the arch of the aorta, is of greater immediate practical significance because of the marked differences in operative mortality than is indicated by the arbitrary anatomical divisions into thirds or fourths.

4. The complication of occlusion of the left stem bronchus due to edema following dissection of the esophagus from the bronchus is reported as occurring in four patients.

5. A case is reported in some detail because of the unusual features and because, in so far as can be determined from the literature, it is the first time a patient has had two successful transthoracic partial esophagectomies.

6. The results of the total operative experience are reported in detail.

The author takes pleasure in expressing his indebtedness to the following surgeons who have collaborated with him on several of the cases included in this report: Dr. Edward A. Cooney, Boston City Hospital; Dr. Charles Sziklas, Dr. Thomas Anglem, and Dr. Hollis L. Albright, Massachusetts Memorial Hospitals.

REFERENCES

1. Torek, F.: First Successful Case of Resection of Thoracic Portion of Esophagus for Carcinoma, Surg., Gynec., & Obst. **16:** 614-617, 1913.
2. Garlock, J. H.: Surgical Treatment of Carcinoma of Thoracic Esophagus, Surg., Gynec., & Obst. **70:** 556-569, 1940.
3. Adams, W. E., and Phemister, D. B.: Carcinoma of Lower Thoracic Esophagus: Report of Successful Resection and Esophagogastrostomy, J. THORACIC SURG. **7:** 621-632, 1938.
4. Churchill, E. D., and Sweet, R. H.: Transthoracic Resection of Tumors of Stomach and Esophagus, Am. Surg. **115:** 897-920, 1942.
5. Garlock, J. H.: Re-establishment of Esophagogastric Continuity Following Resection of Esophagus for Carcinoma of Middle Third, Surg., Gynec. & Obst. **78:** 23-28, 1944.
6. Sweet, R. H.: Carcinoma of the Midthoracic Esophagus, Ann. Surg. **124:** 653-665, 1946.
7. Strieder, J. W.: Discussion of Symposium, J. THORACIC SURG. **13:** 428-429, 1944.

CONGENITAL ESOPHAGEAL ATRESIA AND TRACHEOESOPHAGEAL FISTULA

CLAYTON G. LYON, M.D., AND (BY INVITATION) STANLEY G. JOHNSON, M.D.
SAN FRANCISCO, CALIF.

IN A recent review of the literature, we have been able to find reports of approximately 468 cases of esophageal atresia with or without tracheoesophageal fistula. There has been a renewed interest in the condition and an increased number of successful cases have been reported since the first successful repairs reported by Leven,[1] and by Ladd[2] in 1939.

The anomaly appears to assume several forms classified by Vogt[3] in three main categories and classified under five main types by Ladd (Fig. 1).

Class 1: The upper esophageal segment terminates in a blind pouch in the upper third and the lower esophageal segment is represented by a similar blind pouch, there being no communication with the trachea.

Class 2: The upper esophageal segment communicates with the trachea and the lower segment is a blind pouch.

Class 3: The upper segment terminates blindly and the lower segment of the esophagus communicates with the trachea above the carina.

Class 4: This is similar to *3* except that the lower segment communicates with the trachea at the carina or with the right or left main-stem bronchi.

Class 5: Both segments communicate with the trachea.

In Fig. 1 we have tabulated the types of anomaly as reported in three large series of cases in which the authors have been able to determine that type which existed. In eighty-four cases, seventy-six or eighty-six per cent, were found to have an upper esophageal segment that terminated blindly, and a lower segment that communicated with the tracheobronchial tree by means of a fistulous tract.

The diagnosis is suggested by increased oral secretions, attacks of cyanosis, or the regurgitation of feedings. Confirmation is obtained by passage of a small catheter into the esophagus which meets an obstruction in the upper third, or fails to enter the stomach. The patient is then fluoroscoped while one-half to one cubic centimeter of lipiodol is introduced through the catheter, thus demonstrating the upper segment with or without its tracheal communication. The inferior level of the upper esophageal segment is elevated during the act of deglutition. Therefore, one should determine this level during and between the acts of deglutition under fluoroscopy rather than relying on a roentgenogram for this information. Following the examination, the Lipiodol should be aspirated as it is easily regurgitated and enters the tracheobronchial tree. Roentgenograms of the chest and abdomen are valuable in determining the condition of the lung fields, the presence or absence of other anomalies, and the presence or absence of gas in the gastrointestinal tract. In some instances,

From the Department of Thoracic Surgery, University of California Medical School.

Read at the Twenty-seventh Annual Meeting of The American Association for Thoracic Surgery, St. Louis, Mo., May 28, 29 and 30, 1947.

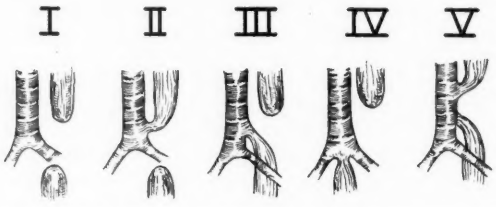
lipiodol which may have been regurgitated by the infant will demonstrate the tracheobronchial tree and occasionally the lower esophageal segment. Gas in the gastrointestinal tract is usually indicative of a fistula. However, Haight records two instances in which the lower segment communicated with the trachea and in which gas was not present in the gastrointestinal tract.

Successful surgical management has been developed and reported by Haight and Towsley,[4] Humphreys,[5] Ladd and Swenson,[6] Gross,[9] Leven,[1] and others. The number of successful repairs is rapidly increasing as a result of earlier diagnosis and constant improvement in anesthesia and surgical management. The early methods of dealing with this anomaly were encouraging but certainly not ideal. The multiple stage procedure with eventual construction of an antethoracic esophagus resulted in the first successful repairs. Subsequently, the development of a plan of direct attack with primary anastomosis of the esophageal segment by an extrapleural approach have proved very successful in those cases in which the esophageal segments are in close approximation. It would appear from a review of the literature that approximately 85 per cent of the cases may be managed by the direct approach. However, there still remains approximately 15 per cent of the cases in which the esophageal segments are so widely separated that the anastomosis cannot be done and in those infants the multiple stage procedure is necessary.

Encouraged by the good results of children undergoing intrathoracic operation under intratracheal anesthesia, and discouraged by the prolonged and difficult course of the multiple-stage operations, we have attempted a transpleural approach under intra-tracheal anesthesia in a small series of cases. In those infants in whom an anastomosis of the esophageal segments is not possible we have contemplated an esophagogastrostomy, fully aware of the fact that such a procedure is one of great magnitude in the newborn infant. In the past six months we have encountered four infants with this congenital anomaly and for whom we have used a transpleural approach under intratracheal ether and oxygen anesthesia.

Ladd mentions the use of the transpleural approach in seven patients with one success, and the successful transpleural ligation of the fistulous tract in one other instance. He also mentions that R. H. Franklin, in 1941, used the transpleural approach in one infant. Haight records one case in which the transpleural approach was used. It has been commonly felt that the transpleural procedure is one of considerable hazard because of the low pulmonary reserve of infants, and that a surgical procedure of this magnitude was attended with considerable shock. It has been also stated that general anesthesia might be less well tolerated and that local anesthesia using the extrapleural approach is preferable. However, many operators using the extrapleural approach have found that the reconstruction of the esophagus is much more satisfactory when using general anesthesia. They feel that there is much less movement of the esophagus and, therefore, less technical difficulty in performing the anastomosis. Furthermore, the pleura is frequently inadvertently opened when the extrapleural approach is used, resulting in pneumothorax.

Intratracheal anesthesia and the transpleural approach theoretically offer certain advantages. With an intratracheal tube in place, the mouth secretions are prevented from entering the tracheobronchial tree. The gastric secretions which enter the tracheobronchial tree before the fistula is tied, can be aspirated frequently to allow for adequate respiratory exchange. Because respiration can be controlled, partial expansion of the lung on the operated side can be maintained. The transpleural approach to the esophageal segments should be easier since the lower segment can be approached below the aortic arch and



LADD 34	3		23	7	1
HUMPHREYS 27	4		22		1
HAIGHT 23	2		21		

Fig. 1.—Classes of esophageal fistula.

developed upward beneath the arch, avoiding, in some instances, a rather tedious search of the posterior mediastinum. The vagus and the recurrent nerves are readily visualized. Ligation of the upper intercostal arteries and mobilization of the arch may be avoided in some cases. With controlled respirations and the abolishment of the act of swallowing, the anastomosis should be technically easier. And, lastly, the divided margins of the pleura and the mediastinal tissues can be sutured to the esophagus, to prevent traction on the anastomosis. The transpleural approach is ideal for the performance of an esophagogastrostomy should it be impossible to anastomose the two esophageal segments.

CASE 1.—R. W., a full-term infant exhibiting typical Mongolian characteristics was admitted to the hospital forty-eight hours after birth. Shortly after birth, attempts to feed the infant resulted in regurgitation and aspiration followed by respiratory difficulty and cyanosis. A catheter could not be introduced into the stomach. Roentgenograms with lipiodol as contrast media revealed a blind upper esophageal segment. The lung fields appeared clear and gas was not seen in the gastrointestinal tract. It was believed that there was no tracheoesophageal fistula.

Because of the Mongolian idiocy, surgery seemed contraindicated. However, after correction of fluid and electrolyte imbalance it was decided to attempt surgical relief.

On Oct. 25, 1946, at the age of 60 hours, the infant was operated upon under ether and oxygen anesthesia as outlined below. A blind upper esophageal segment was found. When the upper segment was mobilized it could be drawn down to a point just above the arch of the aorta. The lower segment of the esophagus was approximately 1.5 cm. long and there was no communication with the trachea. The diaphragm was opened, the vessels of the greater and lesser curvatures were divided down to the prepyloric region, and the stomach was delivered into the pleural space. The lower esophageal segment and a portion of the adjacent lesser curvature 2 cm. in length were excised, and the stomach inverted. The resultant gastric tube could be delivered to a point just above the level of the aortic arch where end-to-side esophagogastrostomy was carried out using two rows of interrupted fine cotton suture. The blood supply to the stomach appeared adequate and there was no undue tension. The diaphragm was closed, its margins were sutured to the stomach, and the site was covered with pleura. The chest wall was closed in layers with fine cotton sutures. The surgical procedure required four hours and twenty-five minutes. At completion of the procedure the infant's condition was considered only fair. Shortly thereafter, a considerable amount of mucoid material was aspirated from the pharynx and one attack of cyanosis occurred. There was no evidence of laryngeal edema.

Because of the Mongolian idiocy exhibited by this infant, the postoperative care was not to be especially energetic. However, on the first postoperative day, the infant received an unplanned feeding of 2 oz. of formula, which was regurgitated immediately and aspirated. Aspiration pneumonia developed and resulted in death forty-eight hours after surgery.

Autopsy revealed that the blood supply to the stomach had been adequate and the anastomosis was intact. The bronchial tree contained a considerable amount of brown mucoid material. There was extensive bilateral pneumonia.

CASE 2.—B. O., a premature female infant weighing 3 pounds and 7 ounces at birth, was admitted to the University of California Hospital 102 hours after birth. At another hospital where she was admitted shortly after a precipitant delivery at home, it was noted that all feedings were regurgitated, that excessive mucus frequently was present in the mouth and nasopharynx, and that attacks of cyanosis occurred with periods of apparent respiratory difficulty. Despite this information, a diagnosis was not established until the fourth day of life, when attempts to pass a catheter into the stomach met an obstruction in the upper esophagus. Thereafter, fluoroscopy revealed gas in the stomach and small bowel; equivocal evidence of pulmonary infiltration on the right; and, with thin barium as a contrast media, a blind upper esophageal segment extending to a level opposite the mid-portion of the third thoracic vertebra.

On entry the infant appeared active but cried feebly. She was markedly dehydrated and moderately icteric. Physical findings otherwise were essentially normal except for equivocal evidence of pneumonitis at the right base. There were no other obvious anomalies.

Several hours were consumed in attempting to improve her general condition. The regime included frequent nasopharyngeal aspirations, continuous administration of oxygen, parenteral injections of plasma, blood, glucose in Ringer's solution, and penicillin.

On Nov. 6, 1946, at the age of 120 hours, under intratracheal ether and oxygen, the transpleural approach mentioned below was carried out. A blind upper esophageal segment was encountered. The lower esophageal segment communicated with the trachea, entering that structure just above the carina. After mobilization of each esophageal segment, the esophageal portion of the fistula was ligated with fine cotton, divided, and the fistulous stump transfixed distal to the ligature with a similar suture. An anastomosis was carried out after rotating the distal segment clockwise through 180 degrees as suggested by Gross. Again attempt was made to take tension off the suture line by suturing the wall of each esophageal segment to the mediastinal tissue. After pleuralization, the wound was closed in layers with interrupted fine cotton sutures.

The operation required one hour and fifty-five minutes, and the infant received parenteral glucose in Ringer's solution and blood during the procedure. At the completion of the operation, her general condition appeared to be no worse than it was on her entry to the operating room.

Postoperatively, however, there was more and more respiratory difficulty, with gradually increasing physical signs of bilateral pneumonia. Associated with these findings were persistent excessive oral and nasopharyngeal mucous accumulations that required aspiration. Death ensued nineteen and one-half hours after completion of the surgical procedure.

Autopsy revealed extensive bilateral pneumonia. The esophageal anastomosis admitted nothing larger than a 2.5 mm. probe, the obstruction apparently resulted from inversion of too much esophageal wall rather than from rotation of the lower segment. Both esophageal segments appeared as though they had been viable, and there was no undue tensions on the anastomosis.

CASE 3.—B. B., a seven months premature male infant, delivered following a pregnancy complicated by placenta previa, weighed 3 pounds, 15 ounces at birth. Physical examination at that time revealed no obvious abnormalities. On the third day, excessive oropharyngeal secretions were noted and the infant regurgitated its feedings through its nose and mouth. This was associated with attacks of cyanosis and difficulty in breathing. At that time examination revealed bilateral, coarse, wet pulmonary râles. A catheter could not be passed beyond the upper portion of the esophagus. Fluoroscopy with lipiodol as contrast media revealed an apparently blind upper esophageal segment. Gas was seen in the stomach and small bowel.

The infant was transferred to the University of California Hospital and prepared for surgery by a regime which included aspiration of the oropharynx, continuous administration of oxygen, parenteral administration of saline, glucose, plasma, and penicillin.

On Nov. 16, 1946, at the age of 88 hours, the infant was operated upon under ether and oxygen intratracheal anesthesia as outlined below. A blind upper esophageal segment was encountered. The lower esophageal segment communicated with the trachea entering that structure just above the carina. Each esophageal segment was mobilized. The lower segment was divided just below its entrance into the trachea, the fistulous esophageal stump was ligated with fine cotton material and transfixed distal to the ligature with similar material. A very satisfactory end-to-end anastomosis between the two esophageal segments was carried out as described below without apparent tension and without rotating the lower segment. The site was covered with pleura and the wound was closed in layers with interrupted sutures of fine cotton.

The procedure required two hours and forty minutes. The condition of the infant at the completion of the procedure was considered quite satisfactory.

During the first seven days postoperatively, the infant was maintained on a regime of frequent oropharyngeal aspirations, continuous oxygen, no oral feedings, and parenteral administration of saline solution, glucose, plasma, blood, and penicillin. During this period the infant seemed to handle its oropharyngeal secretions fairly well.

Thereafter the infant was fed but almost immediately began to regurgitate and aspirate its feedings. Occasionally this resulted in episodes of respiratory difficulty associated with cyanosis. Therefore, feeding attempts were discontinued for a time and the infant was maintained on parenteral fluids, plasma, and blood. On the ninth postoperative day, a small amount of lipiodol was instilled into the pharynx under fluoroscopic vision. The lipiodol was delayed for a short time at the site of the anastomosis and then a narrow trickle passed into the lower esophageal segment and at the same time lipiodol also passed into the left bronchial tree. Apparently the fistula had reopened. It was thought then that dilatation of the anastomotic site might relieve the partial obstruction and it was hoped that the fistula might close spontaneously. Dilatations were done on two occasions until a No. 5 French catheter could be passed with ease into the stomach. However, the infant continued to aspirate its secretions intermittently as well as the small feedings that were given, until pulmonary infection resulted in death on the twenty-seventh postoperative day.

Autopsy revealed evidence that a small abscess had formed about the anastomotic site with resultant reopening of the tracheal fistula. In addition, there was a second area of stenosis of the lower esophageal segment about 2 cm. distal to the fistulous communication of the esophagus with the trachea. This stenotic area was overlooked at the time of the surgical procedure. The lungs showed evidence of bilateral pneumonia and atelectasis.

CASE 4.—P. T., a full-term female infant, weighing 6 pounds, 13 ounces at birth, was admitted to the University of California Hospital twenty-four hours after birth. Shortly after birth an attack of cyanosis was relieved by aspiration of mucus from the pharynx.

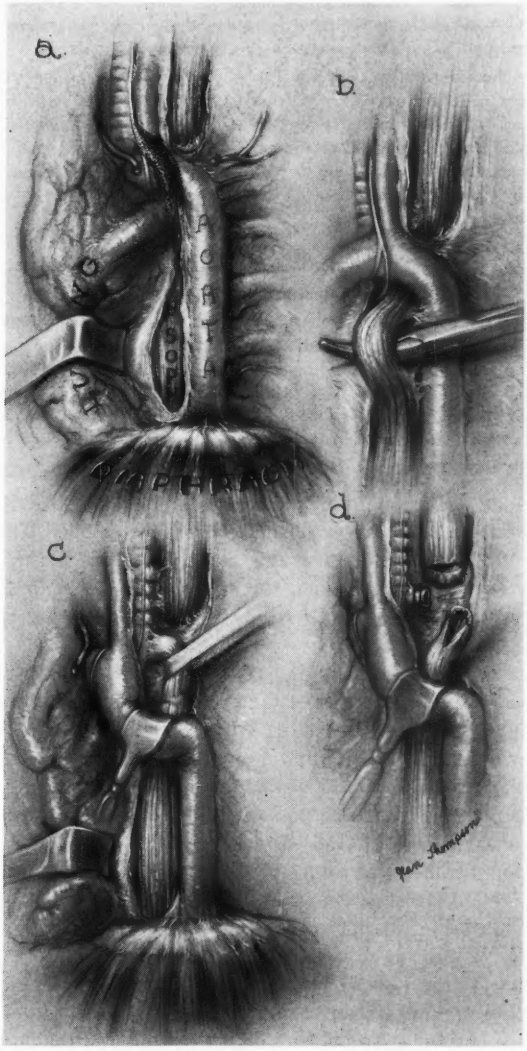


Fig. 2.

It was noted that the child became cyanotic during its first feeding. A catheter was introduced into the esophagus but met an obstruction in the upper third. Physical examination revealed a considerable amount of mucoid material in the pharynx. There were coarse râles throughout both lung fields which cleared somewhat after aspirations of the pharyngeal secretions.

Under fluoroscopy the lung fields were clear. A small amount of lipiodol was introduced into the upper esophagus through a small catheter and it demonstrated a blind upper segment ending just above the tracheal bifurcation. During examination, some of the lipiodol was aspirated and it outlined the trachea and main-stem bronchi. A small amount of the lipiodol was seen entering the lower esophagus through a fistulous tract at the bifurcation of the trachea. Gas was present in the gastrointestinal tract.

The infant's dehydration was corrected, the pharyngeal secretions were aspirated frequently, and twelve hours after entry, at the age of 36 hours, the infant was taken to surgery. Under introtracheal ether and oxygen anesthesia, a left transpleural approach was made. Both esophageal segments were mobilized, the lower segment was freed and doubly ligated at the site of the fistula, and end-to-end anastomosis was effected between the esophageal segments. The procedure consumed three hours and twenty minutes. Ninety cubic centimeters of whole blood were given during the operation.

Following the operation, the infant's condition seemed to be good. During the first forty-eight hours the nasopharynx required aspiration at decreasing intervals. After forty-eight hours the infant appeared to be able to swallow all secretions.

Nutrition was maintained with parenteral blood, plasma, saline solution, and glucose. On the seventh postoperative day the child was started on small hourly feedings of from 4 to 6 c.c. of lactic acid formula. Feedings were taken readily so that they were gradually increased daily until a full formula was taken. Thirteen days after operation the wound healed per primam, and the child was discharged twenty-three days after entry. She is now 7 months of age and in excellent condition. She takes fluids readily but puréed foods must be diluted with formula.

The procedure is diagrammatically represented in Fig. 2. The left posterolateral transpleural approach is employed. The pleural space is entered through the bed of the fifth rib. The third and fourth ribs are divided in the paravertebral area. In the case in which the stomach was delivered into the chest it was necessary to divide the sixth rib as well, in order to gain adequate exposure.

The lower esophageal segment is exposed by a short longitudinal incision through the mediastinal pleura just anterior to the descending aorta. By gentle dissection in the posterior mediastinum, the lower esophageal segment is brought into view. In two infants the lower segment was freed for a distance of 4 to 5 cm. below the arch of the aorta in order to help gain as much freedom from tension on the suture line as possible. This procedure did not appear to jeopardize the blood supply of this segment.

The posterior mediastinum superior to the arch of the aorta is entered by dividing the highest intercostal vein and making a longitudinal incision through the pleura just posterior to the subclavian artery. By gentle dissection, the proximal portion of the lower segment can be freed and delivered above the arch of the aorta, using a small hernia tape for traction purposes. The communication with the trachea is interrupted after ligation of the most proximal portion with fine cotton. The closure of the stump of the fistula is further accomplished by placing fine interrupted cotton sutures.

The upper esophageal segment is then freed as far superiorly in the cervical region as possible. A transverse opening is made in the most dependent portion of the upper esophageal segment. A small catheter is then inserted through the lower segment into the stomach to rule out the presence of additional stenotic areas.

Should none exist, the catheter is then withdrawn. A longitudinal incision of comparable length to the transverse opening in the upper segment is made through the anterolateral wall of the lower segment. It is necessary to use a longitudinal incision in the lower segment because of the progressively decreasing diameter of this segment as it approaches its site of communication with the trachea.

The esophageal segments are then anastomosed using two rows of 00000 Deknatel sutures. Interrupted sutures are used to approximate the mucosa. Mattress sutures are used in the second row to approximate the muscularis.

In one infant in which whip sutures were employed, the lumen at the anastomotic site was found to be very small when examined at autopsy.

An attempt is made to relieve tension on the suture line by placing several sutures from the esophageal wall on each side of the anastomosis to the mediastinal tissues. These sutures also serve to prevent traction on the suture line during the act of deglutition.

From our experience with such few cases we are not justified to draw conclusions. All four infants appeared to have withstood the immediate surgery, even though two were premature infants that were exceedingly poor operative risks. One infant died on the twenty-eighth postoperative day, probably because of the presence of a stricture in the lower esophageal segment and which was overlooked at surgery. In this infant a fistulous tract was re-established between the esophagus and the trachea. The last infant had a very uneventful course. She is well at the present time although she may require dilatations in the future.

REFERENCES

1. Leven, N. L.: J. Thoracic Surg. **10**: 648, 1941.
2. Ladd, W. E.: New Eng. J. Med. **230**: 625, 1944.
3. Vogt, E. C.: Am. J. Roentgenol. **22**: 463, 1929.
4. Haight, C., and Towsley, H. A.: Surg., Gynec. & Obst. **76**: 672, 1943.
5. Humphreys, G. H.: Surg. **15**: 801, 1944.
6. Ladd, W. E., and Swenson, O.: Ann. Surg. **125**: 23, 1947.
7. Daniel, R. A., Jr.: Ann. Surg. **120**: 764, 1944.
8. Haight, C.: Ann. Surg. **120**: 623, 1944.
9. Gross, R. E., and Scott, H. W.: Surg., Gynec., & Obst. **82**: 518, 1946.

DISCUSSION

DR. RICHARD HARWOOD SWEET, Boston, Mass.—There is just one technical matter that I would like to take issue with, namely the technique of tying the esophagus below the tumor and inverting the distal end with a purse string. I have done that several times, under duress, where, because of the poor condition of the patient, I felt it necessary for hastening the operation, but I would like to point out that, at least in my hands, it is very difficult to get an adequate dissection of the pericardial region and the region of the left gastric vessels, which I believe is of great importance if we are going to increase the survival rate in this group.

Might we have the first of three slides which I would like to show to summarize our experience at the Massachusetts General Hospital, which I happened to be reviewing recently?

(Slide.) This represents the experience with 213 patients operated upon with carcinoma of the esophagus, at either the low level, which Dr. Strieder speaks of, involving the cardia, or the high level, in the midthoracic esophagus. Of the latter you can see

there were seventy-two cases. Two of those were inoperable; fifty were amenable to resection and primary anastomosis, which gives us a resectability rate of 69 per cent.

In the lower portion of the esophagus and in the cardia, there were 141 cases, with fifty inoperable and ninety-one resective, giving us a resectability of sixty-four, or about 66 per cent for the whole group.

(Slide.) This gives us the complications and the mortality rate in all cases of carcinoma of the esophagus and cardia treated by resection. In other words, it excludes the inoperable cases, and here we have fifty cases of midesophagus carcinoma treated by anastomosis. Of those, thirty-one made uncomplicated recovery, eight had complications but recovered, and eleven had complications from which they died, which gives us a mortality of 22 per cent for the group operated upon.

In the lower end there were ninety-one, with sixty-two uncomplicated recoveries, eighteen complicated recoveries, and eleven fatalities, with a mortality of 12 per cent.

Now if we do as Dr. Strieder has done with his series, we would take the last group of cases, and during the years 1945-46 there were fifty-three cases in this group. The mortality in the fifty-three cases is 9½ per cent.

(Slide.) This is the last slide. This shows the relative importance—as Dr. Strieder has so wisely pointed out—of differentiating the supra-aortic arch group from the infra-aortic arch group in dealing with the mid-thoracic carcinomas.

In the first of these two groups there were twenty-one cases, and with anastomosis below the aortic arch, fifteen cases were uncomplicated, two were complicated, and four died, which gives us a mortality of 19 per cent.

Now if we take the much more difficult group where the esophagus is pulled up from behind the aortic arch, involving so much more dissection, interference with the thoracic duct and all the other things, we have twenty-nine such cases, with sixteen uncomplicated recoveries, six complicated, seven deaths, and a mortality of 24 per cent.

So if you go from just below the arch to above the arch, there is a difference in mortality of about 5 per cent.

That is the last of the slides. Just one thing more: I am a great believer in this operation as a palliative measure, but we are beginning now to have accumulated enough experience to get some impression about the end results. For example, out of that group of cases which I reported to you, there are twenty-three patients who have been operated upon, over three or more years, so that we can assess the three years' survival rate. Out of that group there are nine who are apparently well.

DR. MICHAEL E. DeBAKEY, New Orleans, La.—I should like to present a technical detail which may be of value under certain circumstances in the performance of esophagogastrostomy, and two cases illustrating its application. This technical detail is concerned with a means of performing the anastomosis in an effort to enlarge the size of the stoma. It was particularly useful in one of the cases which I will show. This was an old lye stricture, and the highest level at which the esophagus could be divided and at the same time provide sufficient proximal length to permit the technical performance of the anastomosis was immediately above the stricture. At this level, however, the esophagus was still somewhat narrowed and had the anastomosis been done using only the cross-sectional diameter of the esophagus, the stoma would have been undesirably small. The problem was readily solved by applying this technical detail, which, incidentally, is an old trick that has been used in other portions of the gastrointestinal tract.

As you will observe from the illustration (slide) the anastomosis is begun in the usual manner by placing the first posterior row of interrupted mattress sutures between the esophagus and the stomach. This layer of sutures is then carried up along the lateral aspect of the esophagus for a distance of about 1½ cm., thus giving an L-shaped appearance to this first posterior row of sutures. The esophagus is then severed just distal to this first row of mattress sutures and the lateral wall incised upward from the distal end paralleling the L-shaped lateral row of mattress sutures. A comparable L-shaped opening is then made in the stomach. The anastomosis is then completed with another posterior row of interrupted sutures for careful approximation of the mucosa and a similar two layers of sutures

anteriorly. The L-shaped opening is considered preferable to the use of an oblique opening in the esophagus because it is less liable to compromise the intramural vascularity than the latter procedure. By means of this L-shaped opening between the esophagus and stomach the final stoma produced is larger than that resulting from the employment only of the actual diameter of the esophagus. Without minimizing the importance of gentle handling and careful approximation of the edges of the esophagus and stomach, especially the mucosal layers, it seems reasonable to assume that by providing a relatively large stoma, the prevention of subsequent stricture formation can be better assured.

The next four slides will show the two cases with high esophageal lesions in which this method of esophagogastrostomy was employed following subtotal esophagectomy. The first patient is a 32-year-old male who, as you may observe from the esophagogram (slide), had a very high stricture of the esophagus, resulting from the ingestion of a solution of lye at the age of 2 years. It is similar to the case recently reported by Dr. Sweet. I should add that in almost every other respect the technique employed was similar to that described by Dr. Sweet. This next slide is a photograph of the roentgenogram following the ingestion of barium taken about 10 days postoperatively. It shows a well-functioning esophagogastric anastomosis, which, incidentally, is actually 16 cm. from the incisor teeth. I doubt that it is possible to make the anastomosis much higher in the chest. The patient is now able to eat and swallow normally.

This next slide shows the preoperative esophagogram in the second case, a 77-year-old Negro with a lesion beginning just about the upper level of the aortic arch that proved on biopsy to be carcinoma. This last slide is a picture of the roentgenogram following ingestion of barium taken on the eleventh postoperative day showing almost the entire stomach in the chest and a well-functioning anastomosis. He was discharged from the hospital in good condition.

DR. RALPH ADAMS, Boston, Mass.—

(Slide.) The first of the two slides shows the over-all results in the cases, with which Dr. Herbert Adams and I struggled. Seventy-one conditions were explored, and thirty-eight resected, with an over-all mortality rate of 34 per cent.

It so happens that we made our radical changes in technique in 1944, and since that time, in twenty-three resections, there have been four deaths, or a mortality rate of 17.4 per cent.

(Slide.) One of the details of technique which helped us greatly, particularly in the prevention of postoperative stricture—I know that Dr. Strieder, from personal talks also uses this, and did not have a chance to mention it in his paper today—is the oblique connection of the esophagus to the stomach.

To further lessen the hazards of postoperative stricture, which, if it occurs, can be dilated over a guiding thread, our policy has been to use two layers of fine, interrupted, nonabsorbable sutures. I should like to commend the traction maintained by the attached tumor or by a right-angled clamp applied above the tumor, on the esophagus while the first part of the anastomosis is being made, which Dr. Strieder emphasized.

It has been my personal preference to remove the spleen, because it is rather in the way, and I have felt that a better anastomosis at a higher level could be more easily made.

Together with Dr. Sweet, I have been anxious to remove as much of the lesser curvature gland-bearing area as possible, and have usually retained only one vessel to supply the stomach, either on the greater or lesser curvature side. The lower third of the lesser left gastric artery is sufficient.

A small point in the closure of the thorax has been of real help in the prevention of fluid accumulations, and that is the use of a Foley catheter, often two Foley catheters. If one introduces it where preference may indicate, and inflates the bulb, it keeps the surface of the lung away from the drainage opening of the catheter very nicely.

Since adopting the Foley catheter, I have had to do a thoracentesis on only one postoperative thoracic resection of any type in over three years, and this has been a matter of comfort. Our longest survival has been fifty months. That patient is now dead.

DR. CAMERON HAIGHT, Ann Arbor, Mich.—Dr. Holinger should be praised for the superb movies which he has shown to us and for the extraordinarily satisfactory technique he has developed for the moving picture demonstration of lesions seen through an endoscope.

The subject of congenital atresia of the esophagus has greatly interested us at the University of Michigan since Robert Shaw in 1938 first called our attention to the possibility of a direct anastomosis of the two segments of the esophagus. Since then we have stressed the restoration of intrathoracic esophageal continuity whenever possible. In fifty-two patients for whom operation has been performed, we have endeavored to apply this principle, even in some cases in which the chance of success was extremely remote. This policy has been used in preference to the alternate method of the multiple-stage operation with an eventual antethoracic esophagoplasty, because we have not felt justified, up until the present time, at least, in recommending an antethoracic esophageal tube as an eventual means of swallowing for a newborn infant.

Our first use of the principle of esophageal anastomosis was in 1939. During the following two years, we operated upon five patients and were discouraged because all of them died. The sixth patient survived, however, and she happens to be the oldest living patient operated upon by this plan and the first one to have survived following the reconstruction of intrathoracic esophageal continuity. She was admitted at the age of 12 days after having been brought a distance of 500 miles by automobile. Subcutaneous glucose infusions had been given en route to prevent dehydration. There was air in the stomach, a sign which we now know is indicative of a good chance for obtaining a satisfactory esophageal anastomosis. Although there was temporary leakage of the anastomosis with the subsequent development of a stricture, it was necessary to dilate the stricture only on one occasion. The slide shows the roentgen appearance of the esophagus in June, 1946, and also a photograph of the patient at the same time. She is a normal child who enjoys normal activities. There is residual narrowing of the esophagus at the site of the anastomosis, but she is able to eat all kinds of foods, although it is necessary that large food particles be omitted from the diet. As there has been progressive enlargement of the lumen at the site of the stricture, further dilatations have not been needed.

The results that have been obtained during the developmental period of the operation of reconstruction of intrathoracic esophageal continuity are shown in the slide. Fifty-nine cases have been seen during the last eight years. Operation was withheld in seven cases; two of these patients were premature, one of them dying twenty-four hours after admission and the other on the second day after admission. Operation was not used in the remaining five patients because of various anomalies in addition to the esophageal atresia.

Operation was undertaken in fifty-two patients. In eleven of the fifty-two an esophagoesophageal anastomosis was not possible; in another patient it was possible, but not done. In these twelve patients no serious attempt was made to correct the anomaly, with the exception of one recent patient for whom an esophagogastric anastomosis was done by Dr. F. X. Byron.

Of 40 patients for whom an esophageal anastomosis was completed, twenty-two died and eighteen recovered, a recovery rate of 45 per cent. The over-all recovery rate in the fifty-two patients for whom operation was undertaken was 34.6 per cent. If the above results are analyzed in groups according to the years in which the patients were treated, it will be seen that the outlook has changed from one of great pessimism at the beginning to one of considerable optimism at the present time. In the first four years of this study, 1939 to 1943, the recovery rate following esophageal anastomosis was 23 per cent. During the last four years, 1943-1947, the recovery rate following esophageal anastomosis has increased to 55 per cent. All the operations have been done within the brief period of eight years and the refinements in management which will largely influence the results in the future have been used only in recent cases. It appears proper, therefore, to analyze further the results in the more recent cases. Thus, in the last two years there have been ten recoveries following esophageal anastomosis in fifteen cases, and within the last three

months six recoveries in six cases. Of these last six cases, three were operated upon by Dr. F. W. O'Donnell, who has the distinction and enviable record of having had a 100 per cent recovery rate to date. We recognize that the nature of the anomaly, the presence at times of associated anomalies and the poor general condition of some of the patients will not allow for 100 per cent recovery of all patients, but the recent results are an index of a figure which can be approximated in the future.

In regard to the type of approach which should be used, we favor the right extrapleural exposure of the anomaly, and particularly if the preoperative roentgenogram demonstrates the presence of air in the stomach, inasmuch as air in the stomach before operation indicates that there will usually be a sufficient length of the two segments to permit the construction of an anastomosis. Thus in forty-four of the fifty-two patients there was air in the stomach before operation and anastomosis was possible in approximately 88 per cent of the forty-four patients. At operation the typical anomaly with a varying degree of separation of the two segments was found in thirty-three of these forty-four cases, and an anastomosis was possible in twenty-eight of the thirty-three. In the remaining eleven of the forty-four patients with air in the stomach before operation, there was partial muscular continuity of the two segments; in this group there was an ample length of the two segments of the esophagus to permit an anastomosis in all cases.

The converse situation is observed when air is not present in the stomach before operation, as was noted in seven of the fifty-two patients. In four of these there was a variable degree of agenesis of the lower esophagus, so that the latter did not extend up to the trachea. An esophago-esophageal anastomosis was obviously impossible because of the short length of the lower segment. In the remaining three of the seven cases, there was a fistulous communication between the trachea and lower esophagus, but the fistula was too small to allow the passage of air. Although an anastomosis was possible in two of these cases, it was unsatisfactory because of the narrow diameter of the lower esophagus.

The excellent opportunity to construct an esophageal anastomosis in patients with air in the stomach, as demonstrated by the above statistics, would indicate that a right extrapleural approach should be favored in this group. In patients without air in the stomach, some other method is needed to obtain recovery in most instances. It would, appear therefore, that in cases without air in the stomach, the left intrapleural approach, which has been used by Dr. Byron in our clinic and recommended today by Dr. Lyon and Dr. Johnson, has great merit, in that it affords an opportunity for the performance of an esophagogastrostomy should an esophago-esophageal anastomosis be impossible, as will usually be the case.

DR. JAMES E. DAILEY, Houston, Texas—For the sake of the record, and because the reported cases of this type of operation are as yet rather small, I should like to offer a small group of only eight cases.

Nine individuals were explored, and resection and anastomosis were performed in eight. One of these operations was done for stricture of the esophagus, two were performed for benign ulcers of the esophagus with marked strictures, one for squamous cell carcinoma and four for adenocarcinoma of the cardia of the stomach extending into the esophagus.

Now if we had stopped with our first five cases, we would have had excellent statistics, as they were 100 per cent recoveries, but the next three died. One of those was a 75-year-old woman with benign ulcers of the esophagus, who died on the third week postoperatively from a peripheral circulatory collapse. An autopsy was performed, and the pathologist could not give us any definite cause of death.

Another of the mortalities was a 71-year-old man, who died twenty-four hours postoperatively, from apparently the same type of circulatory collapse. That was an adenocarcinoma.

The third fatality was a patient 56 years old, who died six weeks postoperatively with an associated empyema.

One of the other patients has since died fourteen months postoperatively from metastasis of the adenocarcinoma to the liver and abdominal organs.

All of these cases were done by the left transthoracic approach. In none of them was it necessary to extend the incision further downward.

I should like to ask Dr. Strieder if he has encountered any troublesome dilatations of the stomach due to sacrificing the vagi.

In all of our cases we have made a habit of not using a Levin tube postoperatively except in two in which we did have some trouble with hugely dilated stomachs. However, the distention disappeared without any further trouble after a few days.

DR. WM. E. ADAMS, Chicago, Ill.—Nine years ago I reported before this association our first case of resection of carcinoma of the lower esophagus. That patient is living and well, without evidence of recurrence today.

Since that time, Dr. Phemister and I have resected approximately sixty cases for this condition, and I would like to report the results on those resections at this time.

During the earlier years, as you all know, the risk of the operation was great for a number of reasons, and our mortality—the same as most—was around 40 per cent. At the time I presented our report nine years ago, the mortality was around 50 to 60 per cent.

You have all heard the statistics given today to show what a marked advance has been made in this short time in the treatment of these lesions. Our over-all mortality in this group of sixty cases is approximately 30 per cent.

In the last year or so, during our last twenty-seven resections, there have been only two deaths. At the present time most of the risk is on a cardiovascular accident basis. There will be an occasional leak, but, as Dr. Strieder has shown us, most of such patients can be brought through with chemotherapy and other means.

I do not quite agree with Dr. Strieder about the mortality being so much greater for the high resection and anastomoses. That certainly has not been true in our hands. I think that is an important factor, too, because so often the lesion is such that we are tempted not to remove enough of the organ, and we are going to have recurrences if we do not make adequate resections.

I also agree with Dr. Sweet, about adequate removal along the cardiac end of the stomach. Our first patient had metastasis to the glands along the lesser curvature of the stomach. If those glands and the tissue in that region are removed, I feel sure that we will have a higher percentage of patients living after a five-year period.

DR. N. LOGAN LEVEN, St. Paul, Minn.—I would like to record my experiences with congenital atresia of the esophagus with tracheoesophageal fistula.

Since 1939, when we had the first successful multiple-stage operation in one of these cases, we have seen forty-two patients with congenital atresia of the esophagus. Of that group there were only three in which no operation was performed. Surgery was refused by the parents of one, one died in the admitting room, and one died twelve hours after admission before any surgery could be carried out.

There were four in which gastrostomy was done as a first procedure. These all died before completion of other procedures. We now know that the primary operation should be within the chest on the fistula, and the gastrostomy, if necessary, may be done as a secondary procedure. There were seventeen patients in which ligation or division of the tracheoesophageal fistula was carried out and the upper blind pouch exteriorized and a gastrostomy performed. Of that group, eleven survived. However, one of these died at the age of 3 years because a gastrostomy tube of large caliber was inserted too far and a perforation of the stomach occurred that was undetected in time for surgery.

We were encouraged by Dr. Haight's first successful primary anastomosis and attempted this procedure. Our first four operations were unsuccessful. The first one was overhydrated with too much saline, and like others we have learned to keep these patients on the dry side. The next three anastomoses leaked, and the tracheoesophageal fistula was reestablished. Since these three cases, we have not encountered this complication of reestablishment of the tracheoesophageal fistula.

Of eighteen patients in which primary anastomosis was carried out, eleven have survived. If we analyze the cases in the past two years, the results have improved considerably.

Twenty-one cases were seen. In two no operation was performed. In five the multiple-stage operation was carried out. Four of these patients are alive and well.

There were fourteen primary anastomoses with twelve successful anastomoses. However, one of these twelve died three months after leaving the hospital of a respiratory infection. This patient had multiple anomalies in addition to the congenital atresia of the esophagus.

I have never encountered a patient where there was a stricture at a point in the esophagus lower than the site of the fistula. This finding is most interesting. I have always passed a catheter into the stomach when I have done an anastomosis. In all of these cases a right-sided extrapleural approach was carried out. I believe that with the extrapleural approach the complications are less severe if there is a leakage at the site of anastomosis. In our department at the University of Minnesota Dr. Vareo also has a 100 per cent record, having operated on two patients with two successful results.

DR. LAWRENCE M. SHEFTS, San Antonio, Texas—I would like to present a case to illustrate some of the technical points that were mentioned concerning carcinoma at the level of the arch of the aorta. The case is that of a 67-year-old man who complained of dysphagia. Barium swallow revealed that the constriction was at the level of the arch of the aorta.

Esophagoscopy revealed that the ulcerating mass started slightly above the superior border of the arch of the aorta. Considering the length of the constriction as seen on the barium swallow, it was evident that with the tumor starting at the superior border of the aortic arch it extended for quite a distance in length along the esophagus. Bronchoscopy revealed that the left main-stem bronchus was pushed slightly forward and slightly to the right. The question arose as to whether or not the left main-stem bronchus was involved in the carcinoma.

In planning an attack on the carcinoma, it was decided that if the left main-stem bronchus were involved and the tumor did not invade the hilar vessels, a left total pneumonectomy would be done along with the necessary surgery on the esophagus.

Fortunately, upon opening the chest, the bronchus came very cleanly off the tumor. In mobilizing the tumor, the first right intercostal artery was torn out of the arch of the aorta. (If one runs into that complication, it can be handled as it was in this case by tying off the first two left intercostals and rotating the arch with the finger on the torn point so that one can reach it to suture it.) The mediastinal pleura was then opened above and below the aortic arch and the enitre esophagus was mobilized, the diaphragm was opened, a clamp was placed across the upper end of the stomach just distal to the cardioesophageal junction, and the esophagus was disconnected from the stomach and brought out over the aortic arch. The upper end of the esophagus was transected above the tumor, the stomach mobilized as has been demonstrated, and anastomosis made anterior to the aortic arch.

In making the anastomosis a circular piece of tissue was removed from the stomach. In spite of this, a very definite stricture resulted which has required considerable dilatation. I am positive that if we had used the technique which Dr. DeBakey described, we would not have had this complication. Dr. DeBakey informed me, after he finished his talk, that since using that particular technique he demonstrated to you in those cases that have gone long enough to ascertain whether or not there would be a possible stenosis, there has been no stenosis.

The first lantern slide shows the typical irregularities noted on barium swallow.

The next lantern slide is a photograph of the specimen and the considerable length of the esophagus which is involved can be readily seen.

The next is a lantern slide with a barium fill-up of the stomach showing the area of anastomosis with the stomach outlined well up above the aortic arch.

The last lantern slide is a photograph of the patient, who is doing quite well.

DR. ALFRED GOLDMAN, Los Angeles, Calif.—I want to take a moment to show a slide and briefly discuss a case that may illustrate the reason why the diagnosis of an esophageal stricture may be made when there is no real stricture.

(Slide.) This is a slide, post mortem, of a baby 5 days old, operated on and dying twenty hours after operation. The death was caused by bilateral pneumonia, which the baby had had two days before the operation.

This slide shows the site of the anastomosis, the operation having been done under a general intratracheal anesthesia. The technique used is that of Gross, using several bites with each suture, tying one over the other to invert the muscularis and thereby bring about the approximation of the mucosa.

The ligated esophageal tracheal fistula is here. It was very difficult to get a catheter all the way in the stomach, and I did not know the reason for that until post mortem.

At this point the esophageal-gastric junction, one sees a very marked thickening in the muscle in the wall of the tube of the lower segment. This muscular hypertrophy is comparable to pyloric stenosis.

I think muscular hypertrophy explains the difficulties in demonstrating the patency of the lower segment, and possibly misdiagnosing it as a stricture.

DR. FRANCIS R. X. BYRON, Ann Arbor, Mich.—I would like to say a few words about those cases with marked hypoplasia of the lower esophageal segment, or with an agenesis of this segment in which the x-ray shows an absence of air in the stomach. I think one is justified in making the presumptive diagnosis of agenesis, and when one combines the x-ray finding with a tracheostomy which shows no fistula between the lower segment and the trachea, one then is justified, I believe, in assuming that the extrapleural direct anastomosis will not be feasible, and that, therefore, a left intrapleural operation and esophagogastrostomy is advisable.

(Slide.) This is a slide that Dr. Haight very kindly loaned to me. There were ten cases in the series at the University of Michigan Hospital in which there was no air in the stomach. Of those, the anatomic defect was proved by operation or autopsy in nine cases. In two of these direct anastomosis of the esophageal segments was possible. In these cases, in spite of the fact that there was no air in the stomach, tracheoesophageal fistula was present, and I think that tracheoscopy is essential to eliminate this possibility.

(Slide.) This baby was admitted at 4 days of age, weighing 5½ pounds. There was no air whatever in his stomach. We elected to do a left transpleural operation.

The upper esophageal segment ended just at the arch of the aorta. It was not mobilized. However, the stomach mobilized readily to reach the level of the aortic arch, so that anastomosis could be made without tension. It does show that the stomach will rise, even in the newborn infant who has never had anything in the stomach.

This is the line of the anastomosis showing excellent healing. This baby lived six days and, to our great disappointment, died late one night. He had had attacks of cyanosis during the postoperative course, and during one of these, the nurse was unable to resuscitate him. The autopsy six days later showed no intrathoracic complications. The anastomosis was nicely healed. What had brought about the baby's death was hypoplasia of both pulmonary arteries with a huge patent ductus arteriosis.

I show this case because it does demonstrate that technically, the operation of esophageal gastrostomy in newborn infants is not only feasible but that that operation is technically much easier than we had anticipated.

DR. W. A. HUDSON, Detroit, Mich.—This is a report on a case that has been reported before this organization previously. It is now approximately eight and one-half years since the patient was operated upon for an extensive carcinoma of the upper third of the esophagus involving also the larynx and upper portion of the trachea.

There was complete resection of the larynx and the first two rings of the trachea, together with the upper third of the esophagus. No attempt was made to reestablish the continuity of the digestive tract. The distal end of the esophagus was attached to the prevertebral fascia in the low cervical region, and the patient has fed herself through a stomach tube passed into the distal portion of the esophagus. She is still alive, and is enjoying good health in spite of the fact that she has been without a larynx or upper third of the esophagus. She travels freely and extensively.

DR. EARLE B. KAY, Cleveland, Ohio.—I would like to discuss briefly the possible advantages of making a tube out of the greater curvature of the stomach for high lesions of the upper thoracic esophagus.

I have had five subtotal esophagectomies where I employed a gastric tube fashioned from the greater curvature of the stomach. To obtain sufficient mobilization of the stomach in high esophagogastric anastomoses it is necessary to ligate the left gastric artery. This supplies the greater portion of the lesser curvature of the stomach. By utilizing the greater curvature half of the stomach in the formation of a tube, the lesser curvature half (that portion devitalized by ligating the left gastric artery) as well as all of the lymph gland-bearing area in this region can be removed. This is a frequent site for metastases. Such a tube will usually reach higher than when the entire stomach is employed. Furthermore, it can be placed in the old esophageal bed underneath the aortic arch. This contributes to less tension on a new anastomosis than when the stomach or gastric tube is brought around the lateral surface of the aortic arch. There is also less tendency to interfere with respiratory function. I would like to inquire as to the type Dr. Strieder employed.

DR. ROBERT R. SHAW, Dallas, Texas.—I would like to add a short word about cases of atresia of the esophagus with or without fistula that do not have air in the stomach.

I agree that this usually means a very atresic lower end of the esophagus. However, yesterday morning my associate, Dr. Paulson, operated on one of these patients, and much to his surprise found there was no tracheal fistula, but that the esophagus was intact all the way except for the atresic area, so that it was a very simple matter to open up the esophagus at the point of the atresia and reanastomose the lumen.

I think, however, if a person encounters a case in which there is no air in the stomach or intestines, that it would be wise to make the left-sided approach, even though, in my experience, the right-sided approach is best for most cases, because this would allow the use of the esophageal gastrostomy technique in case the lower end was atresic.

DR. JOHN W. STRIEDER, Boston, Mass.—With regard to gastric dilatation following these operations, in the resectable cases it has not been an outstanding problem with us. However, in some of the inoperable cases, where both vagi have been divided, we have had troublesome dilatation of the stomach.

The remarks of Dr. Adams and Dr. Sweet relative to the resection of the node-bearing areas were, of course, well made. I neglected to say that in these resections we have attempted meticulous dissection of the nodes above and below the diaphragm, the peri-esophageal nodes and the nodes in the region of the celiac axis. I wish to make it clear that low esophageal cancers are treated like high gastric cancers, namely, with a wide zone of gastric resection.

Regarding the bronchial occlusion which Dr. Kay mentioned, all four of our cases seemed to be definitely due to post-traumatic induration of the bronchus, the tumor having been taken from the region of the stem bronchus, and the endoscopic and x-ray findings were such that it seemed unlikely that it could be due to pressure from dilatation of the stomach. In one autopsied case it was proved not to be due to gastric dilatation.

I neglected to mention, in reporting the patient operated on twice, that, to our mingled feelings of delight and chagrin, a careful histopathological inspection showed no evidence of residual tumor cells.

BRONCHOSCOPIC AND ESOPHAGOSCOPIC CINEMATOGRAPHY

PAUL H. HOLINGER, M.D., GEORGE C. ANISON, M.D. (BY INVITATION), AND KENNETH C. JOHNSTON, M.D. (BY INVITATION)
CHICAGO, ILL.

THE primary value of taking endoscopic motion pictures is to record the view seen by the endoscopist as he explores the air or food passages and to reproduce it on the screen. Each landmark may be identified in turn before proceeding to advance or withdraw the endoscope. The motion pictures show the endoscopic anatomy and the pathology of the passages; the endoscopic appearance of inflammatory processes, tumors, and foreign bodies also can be shown. Pictures have been made which demonstrate the endoscopic techniques used to remove foreign bodies from the air and food passages, the forceps being shown as they actually grasp the coin, tack, button or other foreign body as it is removed. Motion pictures make it possible to demonstrate the living appearance of the air passages with the characteristic changes caused by normal breathing and the pulsations produced by the heart and near-by large blood vessels. These normal movements are also visualized as they occur in the esophagus. Such normal motility of the structures plays an important part in diagnostic techniques, especially in the air passages, since lack of them may indicate and help identify certain abnormal conditions. Motion pictures help the specialist give students and physicians a working knowledge of endoscopic techniques; knowledge far more complete than that obtained by occasionally looking down an endoscope in the bronchoscopic operating room. A realistic sense of perspective is usually seen in endoscopic motion pictures because of the constantly moving lumen which appears to tip at an angle to the endoscope axis. The procedure of scanning the field, showing successive landmarks each in turn, also contributes to this simulated stereoscopic effect.

DESCRIPTION OF CAMERA

The new endoscopic motion picture camera is similar in some respects to a type previously constructed.[1-4] The light-source, focusing telescope, and camera are combined in one unit (Fig. 1). An attaching clamp at the front of the unit permits the instant attachment or detachment of any of several endoscopes or light-reflecting tubes used to photograph the various areas. A heated removable glass slide placed immediately behind the attaching clamp shields the optical parts as the patient breathes or coughs. The apparatus is held by a built-in handle when photographing through the endoscopes, or supported on a tripod for mirror photography of the larynx. The camera is started and stopped by a trigger lever in the handle which also automatically raises the lamp voltage to the proper color-temperature while the camera is running. The plane of sharp focus at the object is adjustable and may be

Read at the Twenty-seventh Annual Meeting of The American Association for Thoracic Surgery, St. Louis, Mo., May 28, 29, and 30, 1947.

placed at any position from 9 to 26½ inches from the attaching clamp. The telescope and camera are focused simultaneously by a knob on the left side of the housing. The telescope shows an exact duplicate of the film image during finding and focusing as well as while the camera is running. A slot permits the insertion of a supplementary lens for photography of the eardrum at a relatively high magnification.

The following description deals with each portion of the camera separately, to show the basic design and theory underlying its operation.

Endoscopes.—The most obvious choice of open-tube endoscope for photography is one which is blackened on the inside to prevent stray light from fogging the picture, or to prevent reflections from the wall of the scope from being included in the picture. However, experience has shown that it is necessary to use endoscopes whose inner surfaces are brightly polished and nickel-plated if sufficient light is to be obtained on the photographic field for taking Kodachrome color pictures. Endoscopes polished on the inside will produce several times the light intensity possible with nonreflecting endoscopes. In a properly designed camera and endoscope, the reflections cause no serious trouble if the finished film is masked with a circular mask to show only the circular field which is visualized through the endoscope. The inner surface of the endoscope near the tip is threaded for a distance of one-half inch which prevents photographing the reflections from this portion of inner surface of the scopes. The out-of-focus images reflected from the remainder of the inner surface are almost completely eliminated by the masking which is described below.

The same endoscope is used both as a bronchoscope and esophagoscope (Fig. 2). It is 16 inches (41 cm.) total length with the free working distance of 11 inches (28 cm.) from the upper teeth. Since the camera lens can be adjusted to photograph at least four inches (10 cm.) beyond the endoscope tip, the total maximum effective free working distance is about 15 inches (38 cm.) for the bronchoscope.

The laryngoscopes are 2½ inches shorter than the bronchoscopes, having 8¼ inches (21 cm.) free working length. This length is more than is necessary for photographing the larynx, but the requirements of the bronchoscopic length determined the focusing range of the lens; this focusing range requires that the laryngoscopes be longer than normal. The camera focus can be adjusted to any point up to 6½ inches (16.5 cm.) beyond the laryngoscope tip. The shorter laryngoscope permits larger pictures on the film than can be made through a tube of bronchoscopic length.

The bronchoscope, small laryngoscope, and esophagoscope are designed to take advantage of a larger diameter of the scope at the upper teeth than that needed to permit introduction into the esophagus or through the larynx. The slightly tapered portion shown increases the light at the tip, compressing the light received to a smaller circle so that sufficient light is obtained at the tip of the scope in spite of its small diameter. Each light ray entering the scope at the large end of the gradual taper is reflected several times before reaching the tip. The steep taper near the attaching flange contributes comparatively

little to the total light; it is used to gather the complete cone of rays from the full diameter of the light source.

The endoscopes are heated briefly in the sterilizer just before use, so that moisture from the air passing through them will not condense on the inside bright surface. If any considerable part of the inner surface becomes covered with droplets of moisture, the light reaching the tip is reduced, and underexposure of the color film results.

The endoscopes have a knurled ring immediately in front of the attaching flange, by which they can be held while they are inserted in the recess of the attaching clamp before the clamp is closed. When re-attaching the camera after removing secretions from the field, the ring allows proper alignment of the scope flange, and quick attachment to the camera. A small raised metal knob on each scope indicates the direction of the tip visually or by palpation.

Attaching Clamp.—The attaching clamp permits instant attachment or detachment of any of several endoscopes or light-reflecting tubes. The clamp makes a solid mechanical connection between the endoscopes and camera unit. The endoscope axis is aligned accurately with the camera axis so that the image of the endoscope tip comes in the center of the camera film aperture, and is approximately centered in the mask opening.

Glass Slide.—A heated glass slide is used immediately behind the attaching clamp to shield the optical parts of the camera from gross soiling or condensed moisture. A rectangular opening at one side of the front face of the glass slide allows passage of air through the endoscope to permit the patient to breathe freely when the bronchoscope or direct laryngoscope is used. The glass is optically flat and introduces no aberration into the film image. It is placed at an angle of five degrees to avoid reflections. After the initial heating of the slide, the heat absorbed continuously from the camera lamp bulb keeps the slide warm enough to avoid fogging.

Illuminating System.—An airplane headlight (landing light) bulb is used as the light source in this camera. It was chosen because its two compact filament coils occupy a very small space about 5 mm. square, and because this lamp can be operated on its side instead of in an upright position. The technical description of this airplane tungsten filament lamp is: 240 watt, 12 volt, 20 ampere; medium prefocus base; C-2 type filament; A-19 bulb (2⅜ inches in diameter); total lamp length 4⅛ inches.

The light from this lamp is directed onto the main axis of the camera by means of two condenser lenses and a plane mirror set across the axis at 45 degrees (Fig. 3). The object image returns through a hole in this mirror. A spherical mirror behind the lamp increases the light level and produces more even illumination. An enlarged image of the lamp filament is projected to a plane about 5 inches ahead of the attaching clamp. This is the optimum distance in order to produce the maximum light level through the endoscopes and light-reflecting tubes.

A heat-absorbing glass is used between the condenser lenses to absorb excess heat from the lamp. This is necessary because the heat reaching the tip of the endoscope is slightly greater than the mucosal surfaces can tolerate

with safety. An additional heat-absorbing glass is used in the ear-speculum attachment to reduce the heat still further when photographing the eardrum. This second heat-absorbing glass has a hole in the center so that the light to the camera lens does not traverse the glass. Both heat-absorbing glasses are of the new high efficiency type recently announced by the American Optical Company; the glass is nearly colorless and absorbs little light.

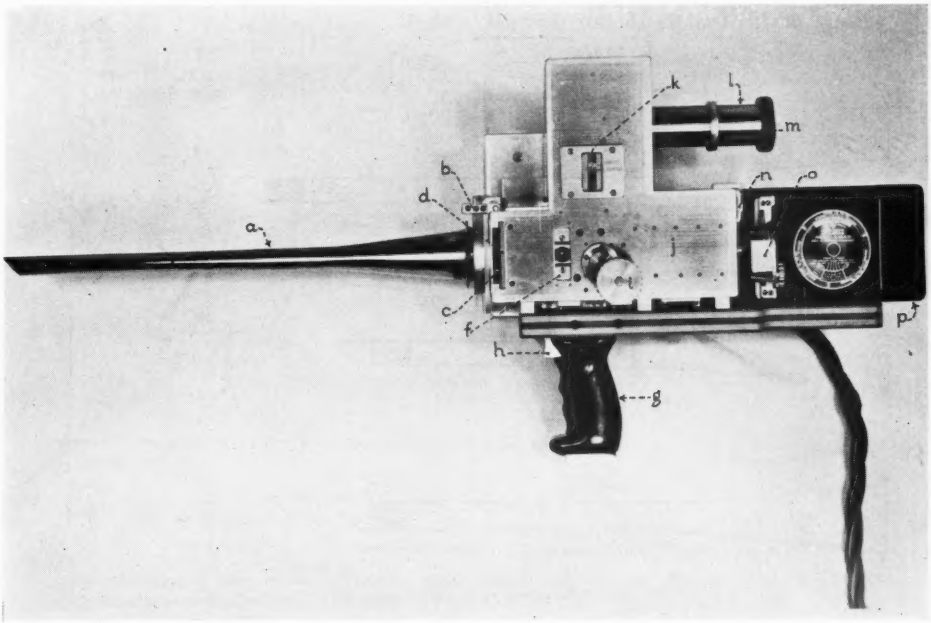


Fig. 1.—Left side view of the camera unit with the bronchoscope attached.

a, Bronchoscope
b, Attaching clamp
c, Opening for breathing
d, Glass slide
e, Lamp house
f, Slot for supplementary lens
g, Handle
h, Trigger
i, Focusing knob
j, Housing for optical system
k, Focus indicator
l, Telescope
m, Eye piece
n, F stop adjusting knob
o, Masking device
p, Bell and Howell autoload 16mm. magazine camera

The lamp is operated at 12 volts while finding and focusing, and is raised automatically to 14½ volts while the pictures are being taken by means of a relay operated by the camera trigger. This produces light of approximately 3450K, the correct color-temperature for type A Kodachrome film. The estimated life of the airplane lamp at 12 volts is about 50 to 100 hours. When operated at 14½ volts, the life is about thirty minutes actual filming time, which permits approximately 750 feet (15 magazines) of film to be taken with one lamp.

A light intensity of about 160 candles-per-square-foot is obtained at the endoscope tip when the lamp is operating at 14½ volts. This corresponds to

a lens setting of f.8 at 1/40 second for type A Kodachrome film. Due to light absorbed in the glass slide and beam-splitter cube, the true stop is about f.6.3 at the camera lens when photographing average-colored areas.

The light intensity at the eardrum through the ear speculum is about 650 candles-per-square-foot, corresponding to a lens setting of f.16 when a lens is focused at infinity. Although some light is absorbed in the beam-splitter cube, and the lens extension ordinarily requires a larger stop, a marked f. stop setting of f. 11 to f. 16 can be used.

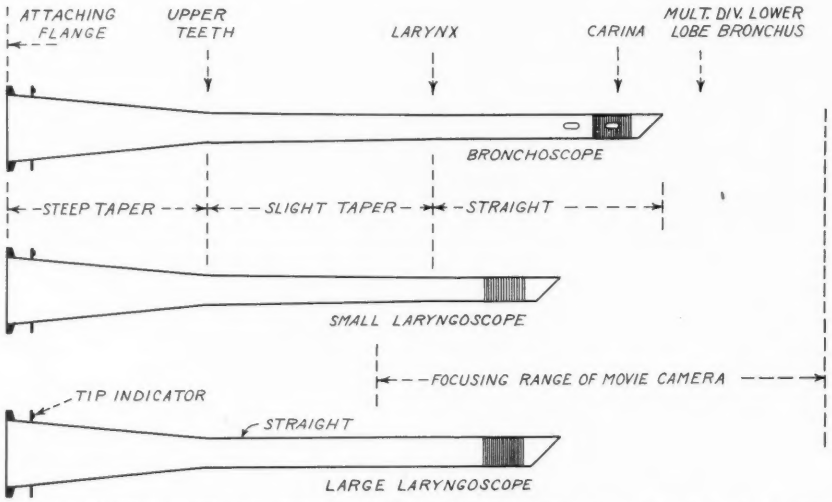


Fig. 2.—Diagram showing construction of the endoscopes. The same tube is used for both bronchoscopy and esophagoscopy.

Safety of Electrical Circuits.—The electrical circuits reaching the camera are at a low voltage and are well insulated from the 115 volt a.c. line (Fig. 4). The low-voltage circuits are insulated from the metal camera housing, and the transformer windings supplying the low voltages are well insulated from the line. With this arrangement, complete safety is provided against electrical shocks to patient or operator.

Optical System.—The basic optical arrangement allows both the camera lens and the focusing telescope to operate on the same axis, and uses the same supplementary lens for higher magnification. Figs. 3 and 5 illustrate the following discussion.

Through the use of a "beam-splitter cube" the camera and focusing telescope view the field at the same time. This beam-splitter cube consists of two right-angle prisms with their hypotenuses cemented together. Before cementing, the hypotenuse of one prism is coated with a very thin partially reflecting aluminum coating. Most of the light reaching the cube passes straight through it to the camera lens. About 15 per cent of the light is diverted at 90 degrees, to the telescope objective lens.

The camera lens and telescope objective lens are identical. They are a matched pair of 90 mm. focal length Wollensak Velostigmats, Series 2. The lenses are of exactly equal focal length, and are of f. 4.5 aperture. They are of the coated type (with antireflection coating) for improved light transmission and improved image contrast. The maximum usable aperture for both lenses is limited to f. 5.6 by the dimensions of the beam-splitter cube, and by the tube through the 45 degree mirror.

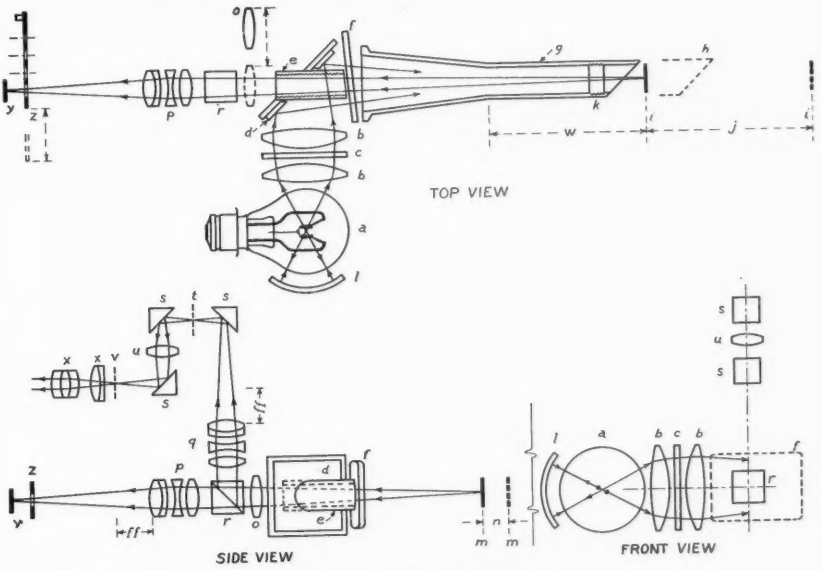


Fig. 3.—Optical drawing of camera (diagrammatic).

a, 240-watt airplane headlight lamp, 12 volts 20 amperes
b, Condenser lenses
c, Heat-absorbing glass
d, 45-degree plane mirror
e, Tube, threaded on inside
f, Optically flat glass slide
g, Laryngoscope
h, Tip of bronchoscope or esophagoscope
i, Plane of sharp focus at object
j, Range of focus adjustment (9 to 26½ inches from flange)
k, Threads on inside of endoscope to produce light ring around image on film
l, Spherical mirror
m, Object plane in sharp focus when supplementary lens "o" is used
n, Range of focus adjustment with supplementary lens (4 to 5 inches from flange)
o, Achromatic supplementary lens, 24.4 cm. (9 ⅝ inch) focal length
p, Camera lens, f.4.5, 90mm. focal length, tessar type
q, Telescope objective lens, f.4.5, 90mm. focal length, tessar type
r, Beam-splitter cube, diverts light to telescope focuser
s, Front-surface mirrors to bring telescope eyepiece to proper position
t, Image plane of telescope objective lens
u, Telescope image-erecting lens. A 1 inch focal length, f.2.5 movie camera lens is used
v, Image plane at eyepiece
w, Free working length of laryngoscope
x, Eyepiece lenses
y, 16mm. motion picture film in camera
z, Movable mask. Four holes shown in top view. (Masks endoscope reflections from film)
ff, Focusing motion of camera and telescope-objective lenses

The two lenses are moved simultaneously by a knob with rack and pinion motion, through equal distances, and without any backlash or play in the gearing. Friction holds the focusing knob wherever it is stopped. Since the focal image planes are fixed in position, the motion of the lenses moves the object plane of sharp focus farther or closer in relation to the attaching clamp, and in this way the camera lens and telescope are always focused upon the same

plane in the object space. The supplementary lens simply shifts both planes of sharp focus a certain distance toward the attaching clamp. With the supplementary lens, the focusing action is basically the same as with the regular endoscopes, but through a shorter distance.

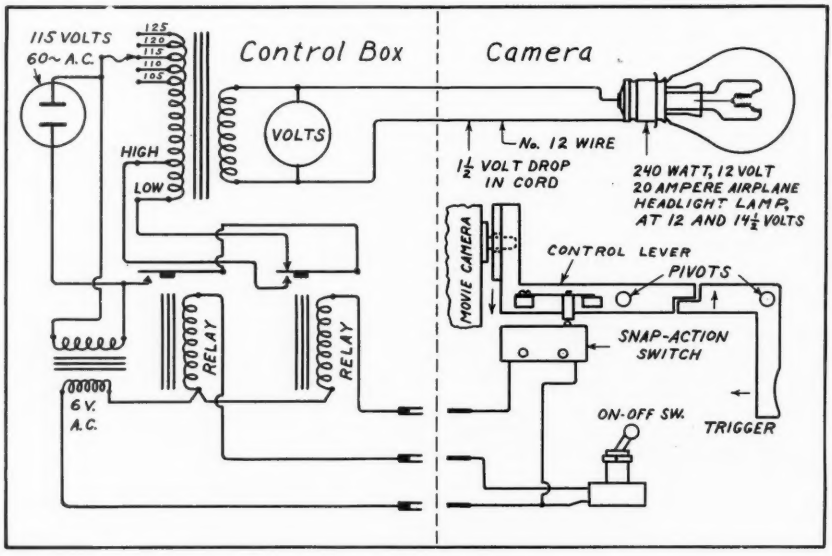


Fig. 4.—Diagram of the electrical system.

Focusing Telescopes.—The focusing telescope is an extremely important part of the camera. By means of the telescope, the field at the endoscope tip is constantly under visual supervision, allowing safe manipulation of the endoscope in order to reach the desired field for photography, or for observation of the field while the endoscope is advanced or withdrawn while filming. The telescope enables the plane of sharp focus for the camera to be placed at any distance beyond the endoscope tip; the plane of sharp focus may be changed while the camera is running. The field is seen at all times, while filming as well as before and after taking the pictures. Experience has shown that the mucosal surfaces of low contrast can be focused upon more accurately by using the aerial image and therefore no ground glass is used at the eye-piece focal plane. The telescope is adjusted to produce correct focus for the camera when the eye is relaxed and focused upon infinity—similar to looking into a microscope. The eye-point is far enough away from the eyecap to permit the operator to wear glasses. The camera is designed for a right-handed person, and for the right eye at the telescope.

The image in the telescope eyepiece is erect and correct from side to side, the same as though the operator were looking directly into an endoscope. The optical system must provide this correct image, since an inverted or transposed image introduces serious risk of trauma when the endoscope is manipulated.

The total magnification of the field as seen in the telescope eyepiece is about five times for the endoscopes, and about ten times for the supplementary lens distance.

A focus indicator is provided in a window at the side of the main housing to show the position of the plane of sharp focus ahead of the attaching clamp. The indicator may be moved by the focusing knob to the position marked on the scale corresponding to the endoscope used. The pre-setting method by means of the indicator is sure and rapid.

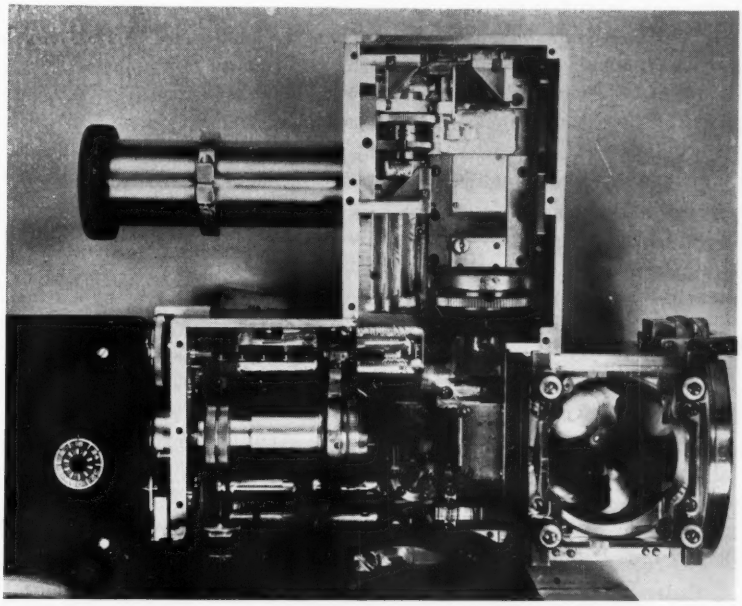


Fig. 5.—Interior of the lens housing viewed from the right side, showing the mechanical arrangement of the paired lenses and the telescope.

Camera Film-Box and Photographic Lens.—The motion picture camera box is a Bell and Howel Auto Load. It is permanently attached to the main housing of the endoscopic camera. The release button is operated by the trigger in the handle. The Auto Load camera uses 16 mm. type A Kodachrome film in 50 foot-pre-threaded magazines and runs about 12½ feet at one winding. This corresponds to 31 seconds of filming time at 16 frames per second. While the normal shutter speed of this camera is 1/43 second at 16 frames per second, the shutter blade opening was increased to allow the light from the lens to remain on the film for a longer period of time, permitting the use of a slightly smaller f. stop. The new shutter speed is 1/34 second. This change is possible because the film image is limited to a circle 0.250 inch in diameter.

The camera model chosen provides for the choice of 8, 16, 24, or 32 frames per second. For bronchoscopic and esophagoscopic photography a speed of 16

frames per second is used at f. 5.6 to f. 6.3. For photography through the laryngoscope a speed of 24 frames per second is used at f. 6.3 to f. 8. The speed of 24 frames per second slows the action of the vocal cords so that their movements seem smoother and more normal in the finished picture.

A special masking device is built into the left side of the camera film box in order to mask off the out-of-focus images reflected from the inside of the endoscopes. The mask is a metal strip with a series of four holes of graduated sizes corresponding to the film image diameter of the various endoscope tips. The largest opening is 0.250 in diameter. The strip is moved by a small knob and the hole selected is centered automatically in the film aperture by a roller which drops into detent notches in the mask strip. The mask is 0.250 inch ahead of the film, and it masks fairly close to the circular film image with only a small margin of the endoscope wall visible in the finished picture. Without such a mask, the bright out-of-focus images reflected from the inside of the endoscopes would distract attention from the circular field of view. With the ear-speculum it is possible to mask down to the exact diameter of the speculum tip.

As previously stated, the camera lens is a 90 mm. focal length f. 4.5 anastigmat type. The maximum usable aperture is f. 5.6. The f. stop is adjusted by a knob at the rear of the main housing, and stops of 5.6, 6.8, 8, 9.5, 11, 13.6 are provided (one-half stop intervals). The maximum circle of confusion on the film, due to aberrations of the lens, is less than 0.001 inch at the film plane. This is entirely adequate for the best quality motion picture photography on 16 mm. Kodachrome film, and is the accepted standard for the best lenses used with 16 mm. cameras.

Depth of Field.—The depth of field (sometimes called depth of focus) at the object plane of sharp focus, is about plus or minus 0.25 inch for a reasonably sharp image. This is for a maximum circle of confusion on the film of 0.004 inch. Theoretically it is possible to show details which are imaged as small as 0.001 inch at the film plane, but 0.002 inch is a good working value for best results. Due to the motion of the screen image, occasional short scenes very slightly out of focus detract little from the general effect. The depth of field values given are adequate and the out-of-focus parts of the image serve to orient the sharp part of the field.

The image on the screen appears slightly enlarged above life size when the pictures are projected. For most of the endoscopic areas the enlargement is about 1.6 life size. This enlargement is based upon the included angle which the image occupies at the eye of the observer. Bronchoscopic pictures are 40 per cent of the screen height, and the large laryngoscopic pictures are 60 per cent. Mirror pictures of the larynx are about 90 per cent of the full height.

Camera Handle.—The camera handle is notched to provide a comfortable and secure grip. The index finger is free to operate the trigger camera release. The handle is located so that the center of gravity of the camera is balanced over the hand when the camera axis is tipped forward to 25 degrees from the horizontal—the approximate angle of the endoscope when the patient is in the recumbent position for direct peroral endoscopy. A threaded hole for a

tripod is located in the bottom of the handle to support the camera while taking mirror pictures of the larynx or for taking pictures at the supplementary distance.

TECHNIQUE

The endoscopes are heated to slightly above body temperature by an electric heating pad or by a brief immersion in the sterilizer just before use. They should be thoroughly dried and their inner surfaces polished. Condensed moisture on the inner surface of the endoscope would reduce the reflection of photographic light from the lamp. The glass slide for the camera is heated for about one minute in the top of the lamp-house; the heat from the lamp keeps it warm after it is inserted in the camera slot. A spare glass slide is available should the slide in the camera become spattered if the patient coughs.

The endoscopes may be introduced into the patient with the camera attached, since the lamp and telescope give full visualization of the field at the endoscope tip. The quick-acting attaching clamp for the endoscopes provides instantaneous release of the camera for aspiration of secretions by suction tube if necessary. In order to observe the endoscopic field when the camera is detached, a light carrier from a standard bronchoscope of proper length is placed in the endoscope. Light-carrier canals and suction tubes are not built into the endoscopes since they would encroach upon the circular field and detract from the appearance of the finished film.

While the endoscope is being introduced the plane of sharp focus is set just beyond the endoscope tip. The moving field is photographed to demonstrate the landmarks seen during the introduction of the various instruments. The plane of sharp focus is then set at a suitable distance ahead of the endoscope tip, and the endoscope slowly advanced or withdrawn with the camera running, in order to show the moving field as seen during an endoscopic examination. In this way, it is possible to take a series of scenes fully recording a bronchoscopic examination.

The location of lesions or obstructions in either main bronchus is easily demonstrated by first photographing the coryna and then proceeding down the bronchus to visualize the actual pathology and its position in relation to the principal bronchial orifices. For orientation in showing esophageal pathology, the scene begins with the introduction of the scope through the cricopharyngeus and into the open lumen of the esophagus. The actual lesion is then photographed; a return to the normal lumen completes the scene.

CONCLUSIONS

A review of the foregoing discussion and of the camera description indicates that motion picture photography through the laryngoscope, bronchoscope, and esophagoscope cannot be done satisfactorily by purchasing an "attachment" for a motion picture camera. In fact, an examination of the equipment described in this article will show that the endoscopic motion picture camera consists of a rather complex optical apparatus which combines the light-source, camera lens, and focusing telescope in one unit, and the "motion picture

camera'' is used only as the film box. The equipment appears very complex, but it is easier and more convenient to use, and takes better pictures of the endoscopic fields than can be obtained with simpler types of apparatus.

REFERENCES

1. Brubaker, J. D., and Holinger, Paul H.: The Larynx, Bronchi and Esophagus in Kodachrome, J. Biol. Phot. Assn. **10**: 83-91, 1941.
2. Holinger, Paul H.: Photography of the Larynx, Trachea, Bronchi, and Esophagus, Trans. Am. Acad. of Opth. and Otol. **46**: 153-156, 1942.
3. Brubaker, J. D., and Holinger, Paul H.: An Endoscopic Color Camera for Otolaryngology and Broncho-esophagology, J. Biol. Phot. Assn. **15**: 73-93, 1946.
4. Brubaker, J. D., and Holinger, Paul H.: An Endoscopic Motion Picture Camera for Otolaryngology and Broncho-esophagology, J. Biol. Phot. Assn. **15**: 171, 1947.

700 N. MICHIGAN AVE.

ESOPHAGEAL HIATAL HERNIAS OF THE SHORT ESOPHAGUS TYPE: ETIOLOGIC AND THERAPEUTIC CONSIDERATIONS

ARTHUR M. OLSEN, M.D., AND STUART W. HARRINGTON, M.D.
ROCHESTER, MINN.

HERNIATION of the stomach through the esophageal hiatus of the diaphragm has been a subject of great interest in recent years. Improvements in roentgenographic methods have enabled the roentgenologist to recognize this condition with increasing frequency. Although the clinician has become conscious of the frequent occurrence of hiatal hernia and is familiar with the symptom complex which suggests this condition, it remains for the roentgenologist actually to demonstrate the presence of hernia.

In 1926, Akerlund[1] classified esophageal hiatal hernias into three types (Fig. 1). In the first type, the esophagus is of normal length and its lower end is not elevated above the diaphragm, but a portion of the stomach has herniated into the posterior mediastinum beside the esophagus. This type has been called "the para-esophageal hiatal hernia" and is the least common of the three types. In the second type of hernia, the esophagus is of normal length but is elevated above the level of the diaphragm and a portion of the stomach is herniated into the posterior mediastinum. This type is by far the most common form of hiatal hernia and frequently is called the "sliding or gastroesophageal type." Actually, the distinction between these first two types of esophageal hiatal hernia is a matter of academic interest only. The mechanism of their origin differs little and the type of herniation encountered is frequently a matter of degree; the para-esophageal hernias frequently progress to form sliding hernias.[2] Likewise, their management is identical, as both types are amenable to a satisfactory surgical repair.

The third type of hernia is the short esophagus with partial thoracic stomach. There is some question as to whether this condition should be considered a true hiatal hernia. Be that as it may, it is extremely important that the hernia which is associated with a short esophagus be differentiated from the esophageal hiatal hernia which is associated with an esophagus of normal length. The management of the two conditions is entirely different, as surgical replacement of the thoracic portion of the stomach into the abdomen can hardly be accomplished by usual surgical methods when the esophagus is short.

The present paper concerns our experience in 220 cases of patients with short esophagus with partial thoracic stomach who have been seen at the Mayo Clinic in the past eleven years (1935 through 1945). As will be readily apparent, hernia of the short esophagus type presents problems different from those of the usual hiatal hernia. It is of interest that the patients with short esophagus comprise approximately 10 per cent of the total number of patients with hiatal hernia seen in this period.

Read at the Twenty-Seventh Annual Meeting of The American Association of Thoracic Surgery, St. Louis, Mo., May 28, 29, and 30. 1947.

From the Divisions of Medicine and Surgery, Mayo Clinic, Rochester, Minn.

PRESENTATION OF DATA

The diagnosis of short esophagus with partial thoracic stomach was made on the basis of roentgenographic findings, esophagoscopic examination, and, in many cases, histologic study of tissue removed at esophagoscopic examination. In many instances the roentgenologic evidence of short esophagus with partial thoracic stomach was indisputable. In 149 of the 220 cases (68 per cent), the diagnosis was confirmed or established by esophagoscopic examination. Difficulty in the differential diagnosis between stricture of the esophagus with and without a partial thoracic stomach was encountered in some cases. In questionable cases, biopsy of the mucosa just below the stricture was taken at esophagoscopic examination for identification of the mucosa. In fifty-two cases of such character, gastric mucosa was recognized at histologic examination. All cases of esophageal stricture of doubtful etiology were excluded from the group.

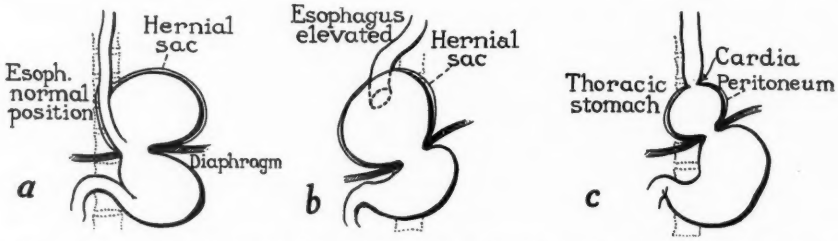


Fig. 1.—Three types of esophageal hiatal hernia; *a*, para-esophageal; *b*, sliding; *c*, short esophagus type.

AGE, SEX, AND OBESITY

Of the 220, 141 (64 per cent) were male and seventy-nine (36 per cent) were female patients. The age of the patients is indicated in Fig. 2. The great majority of patients were in the older age group. In this entire series, 115 patients (52 per cent) were obese at the time of examination or had been obese prior to the development of symptoms of obstruction. On the other hand, twenty-four (11 per cent) were greatly undernourished. These latter also had esophageal obstruction.

SYMPTOMS

The symptoms of esophageal hiatal hernia are many and diverse. Symptoms may be absent or may be severe and disabling. These symptoms may resemble those of any other digestive disturbance or they may present a pattern which suggests the presence of hiatal hernia to the examining physician. Thus, epigastric and substernal distress which occurs after meals and which is intensified when the patient assumes the recumbent position suggests the possibility of hiatal hernia. The distress may be a full feeling, a burning sensation, or pain. It may be referred around the costal margins, into the left side of the thorax, to the scapula, to the neck or arms. The syndrome may simulate the symptom complex of duodenal ulcer, disease of the gall bladder, functional digestive disturbance, intestinal obstruction, or disease of the coronary arteries.

The symptoms of hiatal hernia may be primarily abdominal or thoracic. It is often impossible to distinguish the symptoms of the short esophagus with partial thoracic stomach from those of the paraesophageal or sliding type of hernia. Symptoms referable to the abdomen are much the same in both groups of cases. However, the symptoms arising from the esophagus are particularly significant in cases of short esophagus and have been tabulated in the 220 cases studied in this series (Table I).

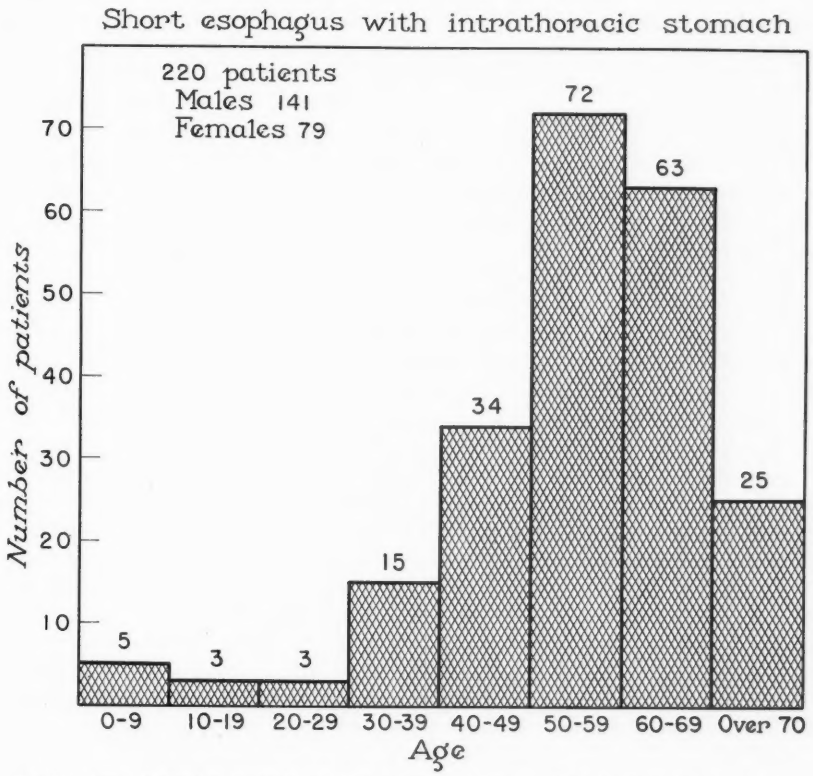


Fig. 2.—Age distribution in cases of short esophagus with partial intrathoracic stomach.

The frequent occurrence of dysphagia is noteworthy. Of the 220 patients, 165 (75 per cent) complained of obstruction to the passage of food on swallowing. In 154 of these patients an actual obstruction was demonstrated, either at esophagoscopic examination or when sounds were passed over a previously swallowed thread. It is of interest that in seventy-six patients, the dysphagia was intermittent or the symptoms of obstruction varied in degree. This observation certainly indicates that spasm of the esophagus plays a definite part in the dysphagia associated with the short esophagus in many cases.

TABLE I. SYMPTOMS REFERABLE TO ESOPHAGUS IN 220 CASES IN SERIES*

SYMPTOM	CASES	
	NUMBER	PER CENT
Obstruction to swallowing	165	75
Substernal distress	71	32
Esophageal pain	20	9
Hemorrhage	18	8
No esophageal symptoms	17	8

*More than one symptom in many cases.

The complaint of substernal burning or distress was noted in seventy-one cases. This symptom was frequently described on the charts as "sour belching" or "acid regurgitation," and patients complained of the burning sensation most often after going to bed. Actual substernal pain was described by twenty patients in the series. In some instances the pain was referred to the neck, shoulders, or arms and had to be distinguished from the pain of cardiac disease. Eighteen patients who did not give any evidence of duodenal or gastric ulcer had had gross hemorrhages. Adequate explanation for hemorrhage from the esophagus usually could be demonstrated at esophagoscopic examination.

ROENTGENOLOGIC DIAGNOSIS

The technique of performing roentgenoscopic examination of the esophagus and stomach in cases of esophageal hiatal hernia has been described by Harrington and Kirklin.[3] The roentgenologic diagnosis of hiatal hernia is exceedingly accurate and is indispensable to the recognition of this condition. In the case of the short esophagus with partial thoracic stomach, special problems are introduced because of the frequency of stenosis of the lower part of the esophagus or associated esophageal spasm. In a number of instances the roentgenologist has requested an esophagoscopic examination to clarify the situation. However, the roentgenologist is able to demonstrate the gastric rugae in the supradiaphragmatic portion of the stomach and to distinguish the intrathoracic part of the stomach from the normal ampulla of the esophagus. The roentgenoscopic diagnoses of our 220 patients are listed in Table II. In 182 of these patients the amount of stomach above the diaphragm was estimated. In 35 per cent there were 2 inches (5 cm.) or less of stomach above the diaphragm. From 2 to 4 inches (5 to 10 cm.) of stomach were visible above the hiatus in 53 per cent, and more than 4 inches (10 cm.) of stomach were intrathoracic in 12 per cent.

TABLE II. ROENTGENOSCOPIC DIAGNOSIS IN CASES OF SHORT ESOPHAGUS (1935 THROUGH 1946)

DIAGNOSIS	CASES
Short esophagus	133
Hiatal hernia	46*
Stricture of lower part of esophagus	30
Esophageal spasm	7
Carcinoma (associated with hernia)	3
Indeterminate	1
Total	220

*In the early years covered by this investigation, no effort was made to determine the type of hernia.

ESOPHAGOSCOPIC FINDINGS

Esophagoscopy was performed in 149 (68 per cent) of the 220 patients. Examinations were performed once in 125, twice in eighteen, three times in four, and four times in two patients. In 126 of the 149 patients on whom esophagoscopy was carried out, the distance from the upper incisor teeth to the esophagogastric junction was measured at the time of esophagoscopy. This distance was 14 inches (35.6 cm.) or more in 36 per cent, and less than 14 inches in 64 per cent. Inasmuch as the distance from the upper incisor teeth to the esophagogastric junction at the diaphragm is approximately 16 inches (40.6 cm.) in the average normal adult, it is evident that the esophagoscopist and the roentgenologist agreed closely in their estimations of the amount of stomach in the thorax.

The presence or absence of ulceration was noted in all cases. Significant ulceration was reported in eighty-nine patients (60 per cent), slight ulceration in twelve (8 per cent) and no ulceration in forty-seven (32 per cent). At the time of esophagoscopy evidence of diffuse ulcerative esophagitis was found in twenty-two (15 per cent).

Specimens of tissue were taken for microscopic examination in eighty-six patients. In many cases esophageal stricture was present and tissue was removed in order to determine the type of mucosa immediately below the obstruction. In fifty-two patients gastric mucosa was demonstrated at histologic examination to prove that a short esophagus was present. In thirty-four biopsy was carried out to exclude malignant disease. In three patients carcinoma was found in association with a short esophagus and partial thoracic stomach. Two of these carcinomas were gastric in origin; the other was a squamous cell lesion. From thirty-one patients inflammatory tissue was obtained. Adenocarcinoma occurring in the herniated portion of the stomach was revealed at transthoracic exploratory operation in one additional case in the series.

Thus it is seen that esophagoscopic examination is extremely important in the diagnosis of the short esophagus with partial thoracic stomach. Esophagoscopy may be necessary to determine the type of hiatal hernia that is present. This is particularly important inasmuch as a patient who has a short esophagus is not a favorable subject for surgical treatment. Biopsy of mucosa from around a stricture is not only desirable in the exclusion of malignant disease, but also is valuable to provide the distinction between cicatricial stricture of the esophagus and stricture associated with short esophagus and partial thoracic stomach. It is our policy to perform esophagoscopic examination in all cases of hiatal hernia in which surgical repair is contemplated and particularly in those cases in which dysphagia or evidence of esophageal obstruction is noted.

In our experience, esophagoscopic examinations are performed most satisfactorily with the aid of both topical and general anesthesia. After the throat is prepared by topical application of a solution of 20 per cent cocaine to the piriform sinuses, the patient is anesthetized with Pentothal Sodium, administered intravenously. In a patient who has a short esophagus, the esophagus

is found to be a straight tube without tortuosity or redundancy. The normal deviation of the lower part of the esophagus anteriorly and to the left is absent. If organic stenosis is not present, the esophagogastric junction is usually found to be open; the normal sphincteric action of the cardia is not encountered, and there is often a free flow of gastric secretions into the esophagus (incompetent cardiac sphincter). Shortness of the esophagus may be demonstrated by actual measurement also.

ETIOLOGY OF THE SHORT ESOPHAGUS

For many years the term "congenital short esophagus" has been used to designate the condition which we are describing.[4-7] In fact, in early articles dealing with the subject, the congenital origin of the condition was taken for granted. In Jackson's early report[8] on this subject and peptic ulceration of the esophagus, the short esophagus was assumed to be congenital. As recently as 1942, Dick and Hurst[4] stoutly defended the conception of a congenital short esophagus. In 1938 Moersch[9] stated that the picture of short esophagus "occurring as a result of cicatrization of the esophagus cannot be dismissed." Allison, Johnstone, and Royce[10-12] have presented evidence to prove that the short esophagus is caused by peptic ulceration of the esophagus followed by healing and contraction.

The basic embryologic and anatomic considerations which concern the esophagus, the stomach, and the esophageal hiatus of the diaphragm have been reported in detail by one of us (S. W. H.)[2, 3, 13, 14] in previous communications. The facts brought out have great bearing on the development of all esophageal hiatal hernias. In brief it should be recalled that the formation of the diaphragm in embryologic life coincides with the descent of the stomach from its thoracic position. The esophageal hiatus is formed by the closure of the diaphragm about the lower end of the esophagus. If the descent of the stomach is delayed, the esophageal hiatus will form about the cardiac end of the stomach. Thus the esophageal hiatus will be abnormally large. In a few instances no further descent of the stomach will occur and the result will be a true congenital short esophagus with partial thoracic stomach. However, in most instances the stomach will continue its descent into the abdomen, thus producing an esophagus of normal length but leaving an esophageal hiatus that is large or incompetent.

The enlarged esophageal hiatus is the probable basis for the development of all types of esophageal hiatal hernias. The frequency with which an enlarged esophageal hiatus can be demonstrated in the course of other abdominal operations has been emphasized by one of us (S. W. H.).[13] The diaphragmatico-esophageal membrane, consisting of elastic and fibrous tissue originating in the fascial coverings of the diaphragm, is the only structure fixing the diaphragm to the lower part of the esophagus. In the presence of an enlarged hiatus, this membrane is hardly strong enough to withstand a traction effect from the esophagus or a pulsion effect from increased intra-abdominal pressure.

All thoracic surgeons are aware of the extreme elasticity of the esophagus. In this connection, the recent studies of Dey, Gilbert, Trump, and Roskelley[15]

are of great interest. They found that stimulation of the vagus nerve in dogs resulted in reflex shortening of the esophagus. The stimulation of the vagus could be a direct electrical impulse or could be indirect, such as would be produced by distention of the gall bladder or manipulation of the liver, stomach, or other viscera. If the thorax in the dog was closed, stimulation brought the stomach up to the hiatus but not into the thorax. If the thorax was opened, the shortening of the esophagus drew the stomach into the thorax. That spasm plays a definite part in shortening of the esophagus, especially in the early phases of the condition, is a fact that has been emphasized by Johnstone,[12] by Smithers[16] and by one of us (S. W. H.).[14] From the work of Dey and his associates,[15] one can infer that associated disease of the upper abdominal organs may be responsible for the spasm and reflex shortening of the esophagus.

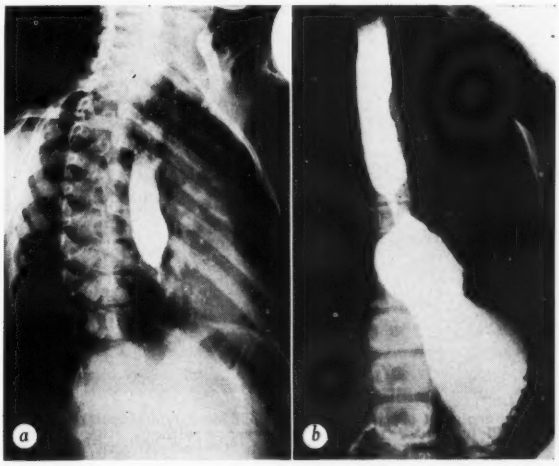


Fig. 3.—Congenital short esophagus with stricture in a boy aged 6 years in 1932 who had had dysphagia since birth. Symptoms have been controlled by dilatations since 1932: *a*, esophagus and stomach in 1932; *b*, in 1945.

Congenital Short Esophagus.—The 220 cases of short esophagus which are the basis of this report were studied carefully from the standpoint of etiology (Table III). Patients who gave a history of dysphagia from birth or early childhood were considered to have a true "congenital short esophagus." There were nine (4 per cent) such patients. Their ages varied from 3 to thirty-six years. Five of them were less than 10 years of age at the time they were first seen at the clinic, two were between 10 and 19 years of age, and two were 35 and 36 years of age, respectively. None of these patients gave any history of ingestion of lye or other caustic poisons. All nine had smooth strictures at the esophagogastric juncture without evidence of ulceration. All were treated successfully at the clinic or elsewhere by means of dilatations performed over previously swallowed threads (Fig. 3).

Why all the patients who had true congenital shortening of the esophagus had associated strictures is a question that is difficult to answer. These cases are similar to the series reported by Findlay and Kelly[5] and Manges and Clerf.[6] Obviously, the presence of esophageal stenosis permitted an early recognition of the condition, and it is conceivable that true congenital shortening of the esophagus has escaped detection in other cases because of the absence of any symptoms.

Fig. 4.—Hiatal hernia of short esophagus type of "indeterminate" etiology in a woman aged 55 years who had had dysphagia for three years without other symptoms. Esophagoscopy revealed a smooth stricture with foreign body (kernel of corn). Dilatations to the size of a 45 F. sound were done on two occasions two years apart.

Short Esophagus of Indeterminate Etiology.—The next group of patients to be considered are those who presented the picture of short esophagus without any obvious etiologic basis. There were forty patients (18 per cent) with shortening of the esophagus of indeterminate etiology. Five of these forty patients, all between 45 and 60 years of age, had no symptoms referable to the gastrointestinal tract. Roentgenologic studies of the esophagus and stomach had been performed at the patients' request. Although no endoscopic studies were done on these five patients, the roentgenologist's diagnosis of short esophagus was definite. The other thirty-five patients in the group in which the etiology was indeterminate came to the clinic complaining of difficulty in swallowing of recent origin but had no other symptoms or history of digestive difficulty (Fig. 4). Facts concerning this group of patients are presented in Tables III and IV. We do not believe that these patients had true congenital, short esophagus. It is obvious that the diagnosis was made at

TABLE III. ETIOLOGY OF SHORT ESOPHAGUS WITH PARTIAL THORACIC STOMACH IN 220 CASES

ETIOLOGY		CASES	TOTAL CASES	PER CENT
I. Congenital: dysphagia since birth			9	4
II. Indeterminate etiology			40	18
A. No digestive symptoms; "accidental finding"		5		
B. Dysphagia: onset late in life and only symptom		35		
III. Acquired etiology			171	78
A. Excessive vomiting		36		
B. Esophagitis associated with hiatal hernia of para-esophageal or sliding type		63		
1. Operative repair	17			
2. Other cases	46			
C. Long-standing digestive distress		57		
1. Duodenal ulcer (without obstruction)	18			
2. Unclassified dyspepsia	39			
D. Scleroderma (acrosclerosis)		9		
E. Antecedent cardiospasm		2		
F. Carcinoma occurring in hernia		4		

TABLE IV. SHORT ESOPHAGUS OF INDETERMINATE ETIOLOGY: DATA CONCERNING THIRTY-FIVE CASES IN WHICH DYSPHAGIA WAS THE ONLY SYMPTOM

AGE (YR.)	CASES	AVERAGE DURATION DYSPHAGIA (YR.)	NATURE OF OBSTRUCTION	
			STRICTURE	SPASM
More than 70	10	4.3	8	2
60-69	13	5.8	8	5
37-59	12	3.6	8	4

a fairly advanced age in this group and that dysphagia developed late in life. This is certainly difficult to explain. However, it is probable that a congenital enlargement of the esophageal hiatus of the diaphragm was present and that atrophy of the diaphragmatico-esophageal membrane took place as these patients grew older.

In many of these cases, the history suggests that esophageal spasm precedes the development of organic stenosis. It seems likely that cardioesophageal relaxation described by Robins and Jankelson,[17] and more recently by Berk,[18] is closely associated with the incompetent or enlarged esophageal hiatus. In cardioesophageal relaxation, the physiologic sphincter at the cardia is incompetent and gastric secretions regurgitate freely into the lower part of the esophagus. We have observed incompetence of this sphincter many times at esophagoscopic examination. As Robins and Jankelson pointed out, the symptom of "heartburn" is not necessarily associated with cardioesophageal relaxation.

Although the etiologic factor in the cases in this group has been labeled as "indeterminate," it is conceivable that a combination of hiatus insufficiency, atrophy of the diaphragmatico-esophageal membrane, spasm of the esophagus, incompetence of the cardiac sphincter, and, finally, ulcerative esophagitis may have produced the end result. The patients probably had an esophageal hiatal hernia of long standing which produced few or no symptoms until cicatrization of the lower part of the esophagus gave obstructive symptoms.

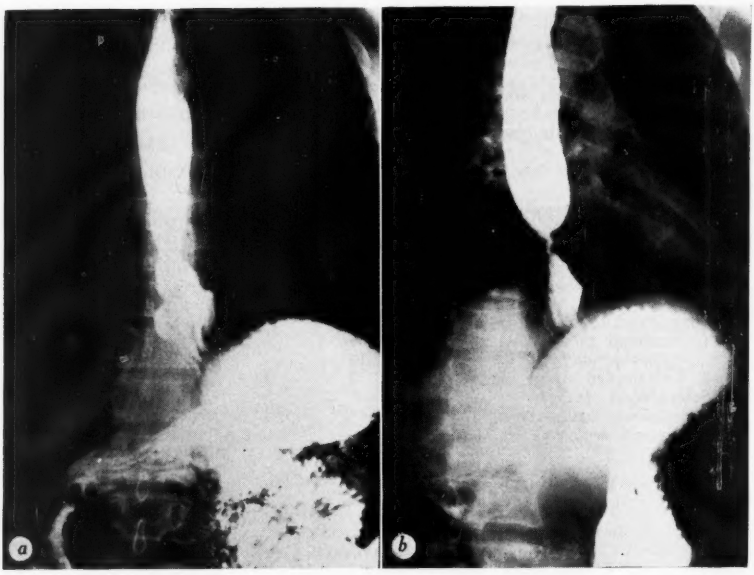


Fig. 5.—Hiatal hernia of acquired short esophagus type due to duodenal ulcer with obstruction in a man aged 58 years who had had epigastric distress for fifteen years but no dysphagia. Esophagoscopy revealed severe esophagitis with ulceration. Ulcer therapy was instituted. In three months marked dysphagia required dilatation and later pyloric obstruction with vomiting and marked esophageal obstruction developed. Gastroenterostomy was performed and stricture at esophagogastric junction dilated three years after initial visit: *a*, 1940, *b*, 1943.

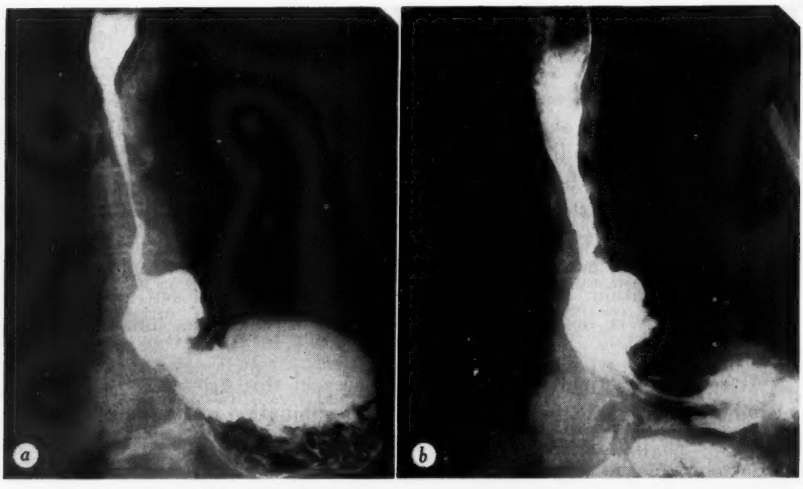


Fig. 6.—Short esophagus with partial thoracic stomach and stricture at esophagogastric juncture, both acquired after resection of the colon for carcinoma, in a man aged 73 years. There was intestinal obstruction with vomiting for two months before operation. Dysphagia (nasal tube had been used) developed two months after operation. Esophagoscopy revealed severe ulceration, esophagospasm, and stricture. Dilatations were needed once a month for relief of dysphagia: *a*, November, 1946, severe ulceration, spasm, and stricture; *b*, March, 1947, after dilatation.

Acquired Short Esophagus.—Of the 220 cases of short esophagus with partial thoracic stomach, there was an apparent etiologic factor in 171 (78 per cent). These cases may be divided into several groups. In the first group the patients gave a history of excessive vomiting. Of the thirty-six patients in this group, nineteen had obstructive duodenal ulcers (Fig. 5), seven had vomiting associated with various surgical conditions (Fig. 6), two had vomiting of pregnancy, one vomited because of a brain tumor, one had frequent bouts of migraine with vomiting, and six vomited as part of a syndrome of dyspepsia or gall bladder disease (Fig. 7). In this group, the stenosis of the lower part of the esophagus was often pronounced and a number of these patients came to us with almost complete esophageal obstruction.

Fig. 7.—Hiatal hernia with short esophagus of acquired type due to gallstones and vomiting in a woman aged 63 years who had had substernal burning and regurgitation of sour fluid for ten years and dysphagia for four years. At esophagoscopy obstruction and ulceration was demonstrated 9¾ inches (24.8 cm.) from upper incisor teeth. Biopsy revealed gastric mucous membrane. Periodic dilatations were carried out.

In the second group of acquired short esophagus there were sixty-three cases (Table III). These patients gave histories and had signs which suggested that an esophageal hiatal hernia of the paraesophageal or sliding type had been present for a considerable time. Characteristically, these patients had experienced epigastric and substernal distress which was made worse by lying down. In this group of cases, it was felt that esophagitis and recurring ulceration of the lower part of the esophagus had resulted in cicatrization and shortening of the esophagus (Fig. 8). Seventeen of these sixty-three patients had had operations for repair of hiatal hernia. In these seventeen patients, temporary interruption of the left phrenic nerve was followed by abdominal operation with replacement of the herniated stomach into the abdomen. In some of these cases shortening of the esophagus was noted at

operation. In others, shortening apparently took place as a result of esophagitis after operation (Fig. 9). The operative cases will be discussed later under treatment.

It is of interest that twenty-three of the 220 patients in our series had gall bladder disease, in addition to the short esophagus with partial thoracic stomach. Also, thirty-two patients gave a history of significant surgical procedures on the upper part of the abdomen exclusive of repair of hiatal hernia.

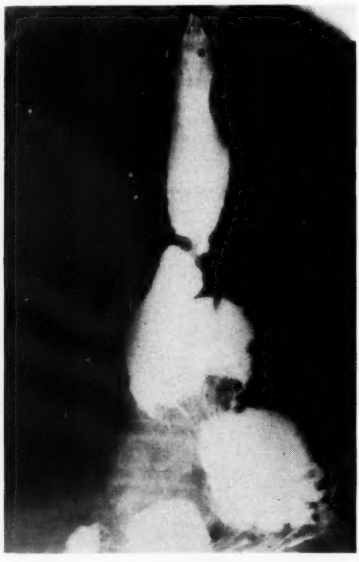


Fig. 8.—Hiatal hernia of short esophagus type in a woman aged 61 years. Diagnosis of large esophageal hiatal hernia had been made ten years previously. Dysphagia was present one year only. At esophagoscopy, marked esophagitis and ulceration were present. Stricture and gastric mucous membrane were 12½ inches (31.8 cm.) from margin of the upper teeth. Relief of dysphagia was by dilatation.

In fifty-seven cases (Table III) the patients gave a history of upper abdominal of long duration. Eighteen of these patients had duodenal ulcers without evidence of pyloric obstruction. The remainder had epigastric distress which was not suggestive of esophageal hiatal hernia.

In nine patients in this series (Table III) a short esophagus with partial thoracic stomach was associated with scleroderma (acrosclerosis) (Fig. 10). The esophageal lesions of scleroderma have been reported by O'Leary, Kirklin, and one of us (A. M. O.).[19] Involvement of the esophageal wall by the sclerodermatous process is apparently responsible for the shortening of the esophagus in this group.

In two cases the short esophagus apparently developed secondary to cardiospasm (Table III). One of these patients, a man 57 years of age, had had dysphagia for twenty-four years. He was seen periodically at the clinic for twelve years before roentgenologic evidence of shortening of the esopha-

gus developed. The other patient, a woman 61 years of age, had both cardiospasm and hiatal hernia in 1941 and on her return in 1944 was found to have a short esophagus.

Four patients were seen who had carcinoma associated with short esophagus (Table III). In three of these patients the carcinoma was gastric in origin (Fig. 11). The fourth patient had a squamous cell carcinoma. Attention is called to previous similar reports by Christiansen,[20] Raven[21] and Smithers,[16] of coexisting carcinoma and hiatal hernia.

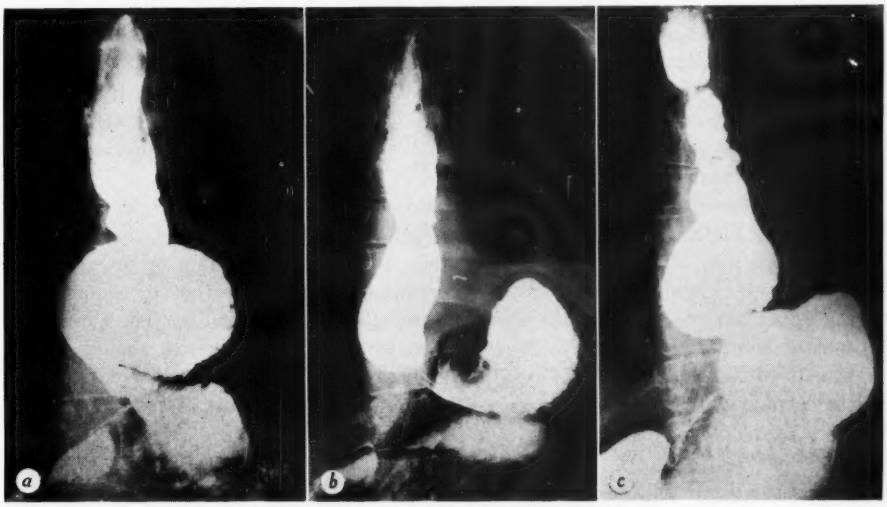


Fig. 9.—Esophageal hiatal hernia with acquired short esophagus due to esophagitis, in a woman aged 60 years who had had epigastric distress for twenty-eight years but no dysphagia; esophagoscopy revealed esophagitis with ulceration at esophagogastric junction 12 inches (30.5 cm.) from the upper incisor teeth. There was a peptic ulcer seen on the left border of the esophagus. *a*, Before temporary left phrenic interruption with repair of hernia; *b*, after operation; *c*, thirty months after operation. Dysphagia had developed. Esophagoscopy revealed stricture 9 inches (22.9 cm.) from margin of upper teeth. Biopsy from region of stricture revealed gastric mucous membrane. Dilatation relieved dysphagia.

Comment.—As has been pointed out previously by one of us (S. W. H.), the evidence indicates that true congenital short esophagus is rare. It occurred in 4 per cent of our group. The congenital factor which appears to play a part in the development of all hiatal hernias is congenital enlargement of the esophageal hiatus of the diaphragm (Table V). Weakening or disruption of the diaphragmatico-esophageal membrane is also necessary for the development of hernia. Atrophy of this membrane apparently occurs in older persons. Increased intra-abdominal pressure, especially that associated with obesity, plays a part in the development of hiatal hernias.

It is reasonably well established that the short esophagus may be caused by severe esophagitis and ulceration of the lower part of the esophagus followed by healing and cicatrization.[10, 11, 16, 22] This is best illustrated by cases of excessive vomiting in which shortening of the esophagus resulted. Acid

TABLE V. FACTORS INFLUENCING THE DEVELOPMENT OF THE SHORT ESOPHAGUS

I.	The enlarged incompetent hiatus
II.	Esophagitis and ulceration of esophagus caused by regurgitation of gastric secretion into the esophagus
	A. Excessive vomiting
	B. Incompetence of cardiac sphincter
	C. Esophageal hiatal hernia
III.	Atrophy or interruption of the diaphragmatico-esophageal membrane
IV.	Reflex spasm and shortening of the esophagus (vagal stimulation?)

gastric secretions undoubtedly produced "peptic" ulceration of the lower part of the esophagus. Yet we have reason to believe that cardioesophageal relaxation alone (incompetence of the physiologic sphincter at the cardia permitting regurgitation of acid secretions into the esophagus) is probably not sufficient in itself to cause esophagitis or ulceration.[12, 16] However, if incompetence of the cardiac sphincter is associated with hiatal insufficiency or hiatal hernia over a long period, ulceration of the lower part of the esophagus with shortening may occur.

Furthermore, reflex spasm of the esophagus appears to play a part in producing hiatal hernia and short esophagus (Fig. 12). Inasmuch as this reflex spasm may be produced experimentally by vagal stimulation or manipulation of upper abdominal organs, it is reasonable to assume that disorders of the upper part of the abdomen should cause this spasm clinically. The correlation of short esophagus with digestive disturbances in our series should add weight to this reasoning. It is probable that reflex spasm and shortening of the esophagus is the first stage in the development of the short esophagus.

TREATMENT

Patients with hiatal hernias of the short esophagus type are not good candidates for radical surgical repair.[2] Results are not nearly as satisfactory as in those cases in which the esophagus is normal in length. Even though it may be possible to replace the stomach below the diaphragm after temporary interruption of the left phrenic nerve, recurrences are common in the short esophagus group. As a rule it is desirable to treat these patients by medical means.

In certain cases, however, surgical treatment of the short esophagus may be indicated. In some cases symptoms are so severe that some type of surgical treatment must be considered. In four patients in our group, temporary or permanent interruption of the left phrenic nerve only was carried out. Three of these four patients were benefited by this procedure; one of them had had severe hemorrhages which did not recur after phrenicotomy.

Seventeen patients in this group underwent surgical repair (Table VI). Abdominal operations were performed, the herniated stomach was replaced, the hernial sac was removed, and the abnormally enlarged esophageal hiatus was repaired. In some cases of short esophagus the surgical problem is that of reconstruction and elevation of the diaphragm to a position above the stomach.[13] In most of our seventeen cases, shortening of the esophagus was noted at the time of operation or had been known prior to operation. In some of the cases,

Fig. 10.—Short esophagus with partial thoracic stomach associated with scleroderma (acrosclerosis).

Fig. 11.—Hiatal hernia of short esophagus type, associated with a carcinoma arising in hernia in a man aged 51 years who had had dysphagia for two months. Esophagoscopy revealed stricture of lower part of esophagus and biopsy revealed adenocarcinoma, grade 4. Dilatations were performed.

however, progressive esophagitis resulted in shortening of the esophagus after an apparent successful repair (Fig. 9).

Clark and Adams[23] described the procedure for transthoracic esophagogastrostomy, which they have employed in cases of benign stricture of the esophagus. This procedure consists in making an anastomosis between the fundus of the stomach and the esophagus above the stricture. This procedure might be employed in cases of short esophagus in which the stricture cannot be dilated satisfactorily.

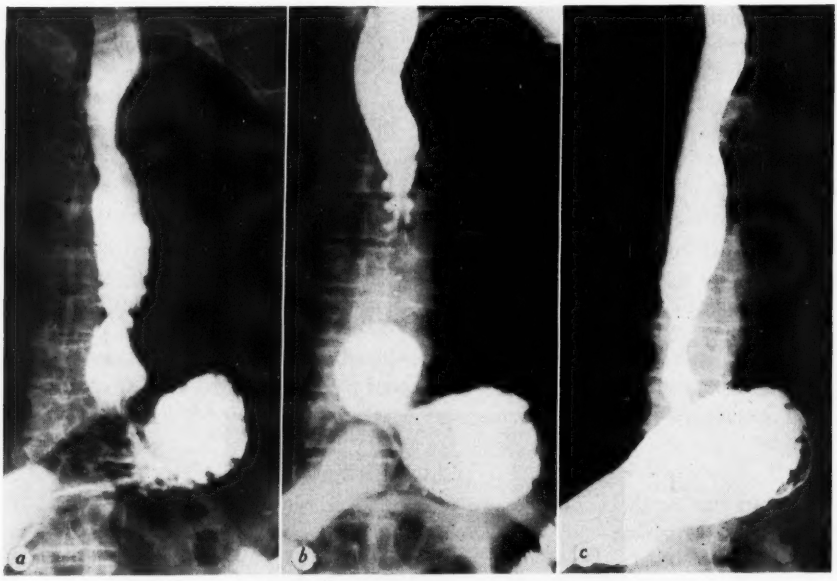


Fig. 12.—Hiatal hernia with acquired short esophagus in a man aged 71 years who had had substernal distress and dysphagia for eighteen months. Esophagoscopy revealed ulceration, and biopsy revealed gastric mucosa. *a*, Ulceration; *b*, spasm played large part; *c*, moderate obstruction to sounds at recent visit three years later.

The most common symptom demanding treatment in the 220 cases was dysphagia. In our experience, dysphagia has been treated most satisfactorily by dilatations performed over a previously swallowed thread. In patients whose dysphagia is caused by spasm, dilatation with a 50 F. sound can be accomplished with ease. However, in the great majority of instances, organic stenosis is present and graduated sounds up to 45 F. must be used. Because of the cicatricial nature of the stenosis, dilatation must be repeated from time to time. Dilatations were performed in 145 of our 220 cases (Table VI). In about half of these only one series of dilatations was carried out at the clinic. In some of the cases twenty or thirty dilatations were necessary in a period of years.

In the early phases of treatment of patients with organic stenosis, dilatations must be carried out every few weeks. However, it usually is possible to increase the intervals between dilatations up to six months or more.

TABLE VI. TREATMENT

TREATMENT		CASES
Interruption of phrenic nerve and surgical repair		17
Interruption of phrenic nerve only		4
Dilatation of stricture		145
Dilated once	79	
Dilated twice	23	
Dilated three times	14	
Dilated four times or more	29	

On the whole, the results of treating these strictures with dilating sounds have been satisfactory. Most of the patients are able to eat almost normally. The risk of performing dilatations in this group by the method described is small. We have had no fatalities resulting from dilatation in these cases. Dilatation should never be done by blind bougienage.[24, 25]

Medical treatment is directed against the further development of ulceration in the esophagus. In the presence of ulcerative esophagitis, a bland type of diet such as that employed in peptic ulcer is desirable. The frequent use of antacid preparations will keep gastric secretions neutralized. For patients who are overweight, a reduction program is imperative. It is amazing how often symptoms of hiatal hernia can be controlled by keeping the weight down and reducing the intra-abdominal pressure. Elevation of the head of the patient's bed is a valuable aid. The head of the bed should be raised 12 to 15 inches (30 to 38 cm.) above the foot. This procedure tends to prevent regurgitation of secretions into the esophagus during the night.

Admittedly, the management of short esophagus with a partial thoracic stomach is not as satisfactory as the surgical repair of the other types of diaphragmatic hernia. However, the majority of patients who have a short esophagus can live comfortably if they will follow the regimen outlined and report for dilatations when necessary.

SUMMARY

A study of 220 cases of short esophagus with partial thoracic stomach was made. On the basis of this review, the following conclusions may be drawn and observations made:

True congenital short esophagus is rare (4 per cent of the cases in our series). In an overwhelming majority of the cases shortening of the esophagus is acquired in later years of the patient's life.

Basic anatomic factors in the development of hiatal hernia are congenital enlargement of the esophageal hiatus of the diaphragm and atrophy or weakness of the diaphragmatico-esophageal membrane.

Shortening of the esophagus develops as a result of "peptic" ulceration of the esophagus. Peptic ulceration of the esophagus may be the result of (1) excessive or prolonged vomiting, or (2) incompetence of the physiologic sphincter at the cardia, when it occurs in association with hiatal hernia or with reflex spasm of the lower part of the esophagus. Reflex spasm of the esophagus in the result of stimulation of the vagus nerve and may be produced by a variety of digestive disorders.

Dysphagia is the most common symptom associated with the short esophagus. In early stages, spasm may be the cause of dysphagia. However, organic stenosis of the lower part of the esophagus develops in most cases.

Hiatal hernia of the short esophagus type is not favorable for surgical treatment. When dysphagia is present, dilatation may be carried out by passing sounds over a previously swallowed thread. Medical measures are directed against further ulceration of the lower part of the esophagus.

Because the treatment of short esophagus type of hernia is radically different from that of the usual hiatal hernia, it is important that an accurate diagnosis be made. Careful roentgenoscopic studies are necessary and esophagoscopic examination is frequently required if hiatal hernias of the short esophagus type are to be differentiated from hiatal hernias with esophagus of normal length.

REFERENCES

1. Åkerlund, Åke: I. Hernia diaphragmatica hiatus oesophagei vom anatomischen und röntgenologischen Gesichtspunkt, Acta radiol. **6:** 3-22, 1926.
2. Harrington, S. W.: Roentgenologic Considerations in the Diagnosis and Treatment of Diaphragmatic Hernia, Am. J. Roentgenol. **49:** 185-196, 1943.
3. Harrington, S. W., and Kirklin, B. R.: The Clinical and Roentgenologic Manifestations and Surgical Treatment of Diaphragmatic Hernia With a Review of 131 Cases, Radiology **30:** 147-156, 1938.
4. Dick, R. C. S., and Hurst, Arthur: Chronic Peptic Ulcer of the Oesophagus and Its Association With Congenitally Short Oesophagus and Diaphragmatic Hernia, Quart. J. Med. n.s. **11:** 105-120, 1942.
5. Findlay, Leonard, and Kelly, Brown: Congenital Shortening of the Oesophagus and the Thoracic Stomach Resulting Therefrom, J. Laryng. & Otol. **46:** 797-816, 1931.
6. Manges, W. F., and Clerf, L. H.: Congenital Anomalies of the Alimentary Tract With Special Reference to the Congenitally Short Esophagus, Am. J. Roentgenol. **33:** 657-669, 1935.
7. Polly, H. F.: Congenital Short Esophagus With Thoracic Stomach and Esophageal Hiatus Hernias; Report of Forty-seven Cases, J. A. M. A. **116:** 821-825, 1941.
8. Jackson, Chevalier: Peptic Ulcer of the Esophagus, J. A. M. A. **92:** 369-372, 1929.
9. Moersch, H. J.: Hiatal Hernia, Ann. Otol., Rhin. & Laryng. **47:** 754-767, 1938.
10. Allison, P. R.: Peptic Ulcer of the Esophagus, J. THORACIC SURG. **15:** 308-317, 1946.
11. Allison, P. R., Johnstone, A. S., and Royce, G. B.: Short Esophagus With Simple Peptic Ulceration, J. THORACIC SURG. **12:** 432-457, 1943.
12. Johnstone, A. S., Harper, R. A. K., McLaren, J. W., and Grossmann, Maria E.: Non-malignant Conditions of the Oesophagus; Symposium, Brit. J. Radiol. **19:** 101-117, 1946.
13. Harrington, S. W.: Esophageal Hiatus Diaphragmatic Hernia, J. THORACIC SURG. **8:** 127-149, 1938.
14. Harrington, S. W.: Diagnosis and Treatment of Various Types of Diaphragmatic Hernia, Am. J. Surg. **50:** 377-446, 1940.
15. Dey, F. L., Gilbert, N. C., Trump, R., and Roskelley, R. C.: Reflex Shortening of the Esophagus in the Experimental Animal With the Production of Esophageal Hiatus Hernia, J. Lab. & Clin. Med. **31:** 499-506, 1946.
16. Smithers, D. W.: Short Oesophagus (Thoracic Stomach) and Its Association With Peptic Ulceration and Cancer, Brit. J. Radiol. **18:** 199-209, 1945.
17. Robins, S. A., and Jankelson, I. R.: Cardio-esophageal Relaxation, J. A. M. A. **87:** 1961-1964, 1926.
18. Berk, Morris: Cardio-esophageal Relaxation, Gastroenterology **5:** 290-298, 1945.
19. Olsen, A. M., O'Leary, P. A., and Kirklin, B. R.: Esophageal Lesions Associated With Acrosclerosis and Scleroderma, Arch. Int. Med. **76:** 189-200, 1945.
20. Christiansen, H.: Quoted by Smithers, D. W.[16]
21. Raven, R. W.: Thoracico-abdominal Gastrectomy for Carcinoma of Thoracic Stomach, Brit. J. Surg. **29:** 39-46, 1941.
22. Huber, Frank, and Andreson, L. H.: Gastric and Duodenal Ulcer Associated With Lesions of the Lower End of the Esophagus and the Cardia of the Stomach, Am. J. Roentgenol. **48:** 158-162, 1942.
23. Clark, D. E., and Adams, W. E.: Transthoracic Esophagogastrostomy for Benign Strictures of the Lower Esophagus, Ann. Surg. **122:** 942-953, 1945.

24. Vinson, P. P.: Treatment of Dysphagia from Hernia Through Esophageal Hiatus in Diaphragm, Arch. Otolaryng. 38: 27-31, 1943.
25. Benedict, E. B.: Benign Stricture of the Esophagus, Gastroenterology 6: 328-329, 1946.

DISCUSSION

DR. STUART W. HARRINGTON, Rochester, Minn.—This study of 220 cases of short esophagus with elevation or herniation of the stomach through the esophageal hiatus was made to obtain criteria for the establishment of the clinical diagnosis and classification of the different types that occur. These data are essential in order that those types that are most effectively treated by conservative measures and dilatation can be distinguished from those that demand surgical treatment as well as from those that require both dilatation and surgical intervention.

I have been chiefly interested in the types of short esophagus that do not respond to conservative management, and in which surgical treatment has to be considered. I would like to emphasize some of the important points made in this presentation: (1) that dysphagia and hemorrhage in cases of esophageal hiatus hernia usually indicate a complicating lesion of the esophagus that must be carefully investigated before surgical treatment is instituted, and (2) that roentgenographic studies may not reveal an associated lesion of the lower part of the esophagus because the lesion may be obscured by the barium in the hernial sac. Roentgenograms may also indicate the presence of a short esophagus of the congenital type when, in reality, the esophagus is not actually short but appears so because of the contraction of its longitudinal muscles, which elevates the cardia above the diaphragm due to an incompetent hiatus. The third point which I wish to emphasize is that an esophagoscopic examination is essential in all cases of hiatal hernia in which surgical treatment is being considered, because it is the most accurate method of determining whether there is any complicating lesion of the esophagus or cardia.

In considering cases of short esophagus from a surgical standpoint, it is important to differentiate them from true hiatal hernias because their surgical management is entirely different.

In cases of true hiatal hernia, the surgical problem is that of replacement of the herniated portion of the stomach into the abdomen, removal of the hernial sac, and repair of the muscle ring of the abnormally enlarged esophageal hiatus.

In cases of short esophagus of the congenital or acquired types, the surgical problem is that of elevation of the diaphragm and the crural fibers of the hiatal muscle ring to a position above the stomach, and the reconstruction of the hiatal muscle ring around the lower part of the esophagus at least 2 to 3 cm. above the cardia, because there may be some contraction of the longitudinal muscles of the esophagus after the operation.

Patients who have esophageal hiatal hernias of the short esophagus type often have associated stenosis of the esophagus for which dilatation should be performed before operation. These patients should be examined at frequent intervals after operation, because they may require more dilatations, depending on the subsequent course.

Great care should be used in the selection of these cases for surgical treatment, because of all the different types of esophageal hiatal hernia, the short esophagus type is the most likely to recur. Surgical treatment of elevation and reconstruction of the hiatal muscle ring should be considered in those cases in which a moderate shortening of the esophagus is present, and in this select group, conservative treatment should be tried before operation is done.

In some cases, interruption of the phrenic nerve and dilatation of the esophagus may relieve the symptoms. Patients who have severe symptoms that do not respond to conservative treatment and the usual operative reconstruction of the hiatus may require the more radical surgical treatment of transthoracic and transdiaphragmatic resection of the esophagus with esophagogastro-anastomosis.

DR. RICHARD H. SWEET, Boston, Mass.—Dr. Olsen made one statement which interested me a great deal. I have been puzzled by this and very much impressed by the

clinical entity which he has pointed out. He mentioned that in one of his cases he thought the etiology was the presence of a carcinoma in the lower end of the esophagus, if I understood him correctly.

Of the 91 resections of carcinoma of the cardia, some of them arising in the stomach and some of them from the esophageal side—which I reported a while ago—exactly 13 per cent were present in a short esophagus and hiatus hernia syndrome. That seems to me to be a very high percentage, and I wish that he might comment upon it from the etiological standpoint.

DR. H. BRODIE STEPHENS, San Francisco, Calif.—The etiologic factors concerned with the production of the ulcerative and ultimate stricture in the group of patients presented by Dr. Olsen are particularly puzzling to me. It seems to me that there are certainly the same factors present in both varieties of hiatus hernias, yet the stricture is only present in the short esophagus type of hiatus hernia.

It is possible that the herniated portion of the stomach in the short esophagus type functions somewhat as a blind loop with the development of high acid concentrations in the portion of the stomach above the diaphragm. Such high acid values could account for the ulceration and stricture that is common to this type of hiatus hernia.

DR. CAMERON HAIGHT, Ann Arbor, Mich.—I would like to present for Dr. Alexander the case of a child of 4½ years of age whom he treated because of excessive bleeding from ulceration of the lower portion of a congenital short esophagus. We have had two such cases; in the second one it was possible to control the symptoms by antacid therapy combined with a temporary phrenic nerve interruption.

The first patient was admitted because of a profound anemia. The symptoms consisted of abdominal pain, occasional vomiting, and inability to take any food except milk. The hemoglobin on admission was 4.35 Gm. and the red blood cell count was 1.3 million. The vomitus at times was reddish brown in color and all stools showed a 4+ guaiac test. After many transfusions the hemoglobin increased to 11.8 Gm. but because of continued bleeding in spite of antacid and dietary management, further measures were required. To the best of our knowledge, esophagogastrostomy had not been employed previously for this type of lesion in a young child, and the case presented the particular problem of the advisability of esophagogastrostomy in a child of this age. As there appeared to be no alternative, Dr. Alexander proceeded in October, 1945, with resection of the lower esophagus and upper portion of the stomach followed by esophagogastrostomy.

(Slide.) The first roentgenogram shows the typical finding of a short esophagus which was presumed to be congenital because of the age of the patient. The second roentgenogram shows the appearance of the esophagus and stomach thirteen months after the esophagogastrostomy.

The child's condition was poor before operation. At operation the lower esophagus was found to be indurated and pathologic examination of the resected specimen showed the presence of a chronic ulcer. The postoperative convalescence was stormy and feedings were not tolerated well. A gastrointestinal roentgenogram showed the greater portion of the stomach to be within the left hemithorax, but there was no evidence of esophageal or pyloric obstruction. In view of the symptoms, a jejunostomy was performed one month after operation. His condition then very gradually improved and the jejunostomy tube was removed four and one-half months later. The patient was discharged from the hospital six months after the esophagogastrostomy.

Subsequent examinations on four occasions at intervals between nine and eighteen months after operation show that the patient is progressing unexpectedly well. He is eating all types of food, gaining weight and strength, and enjoying normal activities. There has been no recurrence of the bleeding. The postoperative roentgenogram thirteen months after operation shows essentially the same appearance of the stomach that was noted before the jejunostomy.

This case is mentioned because of the profound bleeding which resulted from the lesion, the use of esophagogastrostomy in the management of the problems and particularly the early age of the patient at the time of the operation.

DR. WM. E. ADAMS, Chicago, Ill.—We have had a number of these cases, most of them responding quite well to the treatment presented by Dr. Olsen, namely, repeated dilatation and medical measures. I wish to present some slides of a case that I saw about a year ago that presented a different problem.

(Slide.) This was a Mexican boy of 17, who had difficulty in swallowing shortly after birth. It was noted that he had some stricture. He was dilated repeatedly, but at the age of 5 a gastrostomy was necessary. Since that time he led a fairly normal life, developed normally, and when I first saw him, this x-ray showed a complete obstruction at the lower part of the middle third of the esophagus.

Because of some experience with gastroesophagostomy for benign stenosis in other cases, I decided that it was the best procedure in his case.

(Slide.) Through an incision in the lower left chest, extending down in the abdomen, we brought the fundus of the stomach up after releasing and closing the gastrostomy and dividing the left gastric artery. It was relatively easy then to unite the esophagus above the point of obstruction with the fundus of the stomach by a side-to-side operation, very similar to a gastroenterostomy.

He tolerated the operation very well and, as you see here, the obstruction was completely relieved. Since that time he has gotten along very well.

(Slide.) The next slide shows the scar of the operation, extending down into the left upper quadrant of the abdomen. In patients where there is a high degree of obstruction, where dilatation has to be repeated frequently and gives less satisfactory results, I think an operation such as this type is useful.

DR. A. M. OLSEN, Rochester, Minn.—Dr. Sweet has asked whether a carcinoma which occurs in conjunction with the short esophagus is the cause of the shortening of the esophagus, or whether the two conditions occur coincidentally. This question is very difficult to answer. Carcinoma of the cardiac end of the stomach often involves the outer walls of the esophagus. It is our impression that invasion of the lower part of the esophagus by a gastric carcinoma may result in shortening of the esophagus. There is no reason, however, why carcinoma cannot arise in the shortened esophagus or the intrathoracic portion of the stomach.

Dr. Stephens' question concerns the development of stenosis of the lower part of the esophagus in patients who have esophageal hiatal hernia. The cardiac sphincter often is incompetent in the presence of hiatal hernia. Gastric secretions regurgitate into the lower part of the esophagus and may cause severe esophagitis. As this esophagitis heals, contraction and stenosis of the lower portion of the esophagus is likely to occur.

I am very glad that Dr. Adams could discuss his experience with esophagogastrostomy for strictures associated with esophageal hernia in the presence of a short esophagus. Reference to the work of Clark and Adams is made in the text of this paper. Although we feel that most of these strictures can be treated successfully by means of dilatation, there are certain cases in which a surgical procedure, such as that described by Dr. Adams, would be most valuable. I was interested particularly in the patient with esophageal stricture associated with scleroderma who was treated by esophagogastrostomy.

RESECTION OF A COARCTATION OF THE AORTA WITH SUBCLAVIAN-AORTIC ANASTOMOSIS

Report of a Case With a Discussion of a New Clamp for Aortic Resection and Anastomosis

H. H. Bradshaw, M.D., J. F. O'Neill, M.D. (by invitation), and Felda Hightower, M.D. (by invitation)
Winston-Salem, N. C.

THE signs and symptoms of coarctation of the aorta form a well-known symptom complex that needs no repetition now. The diagnosis is not difficult if the possible existence of the lesion is considered.

The prognosis in patients afflicted with this deformity is rather poor unless the patients are treated surgically, and approximately 75 per cent of them will die before the age of 40 years. Hines and Christensen[1] have listed three types of events which may lead to death. First, the condition may remain latent throughout life and death may occur suddenly from a ruptured blood vessel or from conditions unrelated to the vascular anomaly. Second, cardiac insufficiency may occur rather suddenly in an apparently healthy individual and progress to complete decompensation and death. Third, chronic cardiac insufficiency may lead to cardiac failure and death. Subacute bacterial endocarditis and aortitis may also occur.

In view of the poor prognosis in untreated patients, and the encouraging results which have been secured by Crafoord and Nylin,[2] Gross,[3] and Clagett,[4] it is reasonable to advise patients with this deformity to have surgical correction.

Apparently, the first attempts to apply experimentally obtained experience in the surgical correction of coarctation of the aorta was made by Crafoord and Nylin[2] in October, 1944. At that time, two patients were successfully operated upon. Since then Gross[5] has operated upon at least sixteen patients with two deaths; Crafoord[2] has completed two such operations, and Clagett[4] has performed one at last available report. In some of these patients where the coarcted area is rather long, difficulty may be experienced in anastomosing the ends of the aorta after resection of the narrowed portion. It may not be possible to bring the aortic ends together at all, or, if they are approximated, the tension required to maintain them may cause the sutures to break or tear through the aortic wall, especially if it is atheromatous. In such a situation, the procedure used experimentally by Blalock and Park[6] may prove valuable, that is, anastomosis of the proximal end of the divided left subclavian artery to the distal end of the aorta after resecting the coarctation. This method was used by Blalock and Park[6] on a series of forty-three dogs. In each the aorta was severed at the level of the obliterated ductus and both ends were sutured. The left subclavian artery was then divided and its proximal end sutured to the aorta

From the Department of Surgery, Bowman Gray School of Medicine of Wake Forest College, and North Carolina Baptist Hospital, Winston-Salem, N. C.

Read at the Twenty-seventh Annual Meeting of The American Association for Thoracic Surgery, St. Louis, Mo., May 28, 29 and 30, 1947.

by an end-to-side anastomosis. Ten of these animals survived for several months or longer. The predominating complication was paralysis of the posterior part of the body.

These results, although not too encouraging, do not mean that this procedure is contraindicated in patients who have coarctation, and where the aortic ends cannot be approximated after the coarctation is resected. This is true for two reasons. First, patients with coarctation may well have a left subclavian artery which is dilated to the same caliber as the aorta below the coarctation, and it could thus shunt a great deal more blood to the distal aorta than could an undilated left subclavian artery in an otherwise normal dog. Second, even if the left subclavian artery were somewhat smaller in diameter than the aorta distal to a coarctation, it would still be larger than the area of the coarctation itself, and the other collateral channels already present could play an important part in transporting blood to the lower two-thirds of the body.

Resection of aortic coarctation with subclavian aortic anastomosis has been performed on two human beings. Clagett,[4] in August, 1946, carried out this procedure on a 34-year-old man and the patient survived. A similar operation was done on the patient herein reported, but death ensued twenty-four hours after operation from pulmonary edema and atelectasis. In neither patient was there any suggestion after operation of any paralysis in the legs or left arm.

The patient operated upon by Dr. Bradshaw was a 16-year-old white man admitted to the North Carolina Baptist Hospital on Dec. 16, 1946. Three months prior to admission he had noted the onset of dizzy spells when he would rise to his feet from a sitting or lying position. At the same time he noted the onset of frontal headaches, pain in his anterior chest to the left of his sternum, dyspnea on exertion, and some pain and tenderness in the anterior muscles of his thighs. He had also complained for a long time of cold feet. He had consulted three physicians at various times and was told he had high blood pressure. The fourth physician, after an examination which included a chest x-ray, told him that he had a disorder of a large blood vessel in his chest.

When a younger boy he had had frequent nose bleeds and sore throat. An appendectomy had been done in 1944, and several months prior to admission he had been treated in another hospital because of impairment of renal function. At that time, his blood nonprotein nitrogen was reported to have reached a high of 260 mg. per cent. When examined at the time he came to the hospital, there was slight fullness of the veins of his neck. Systolic blood pressure readings in the right arm averaged 142 mm. of mercury, and in the left arm between 136 and 152 mm. of mercury. Diastolic blood pressure in the right arm averaged 70 mm. of mercury, and in the left arm varied between 64 and 70 mm. of mercury. In the legs, the systolic blood pressure readings varied between 84 and 108 mm. of mercury, while the diastolic readings varied between 60 and 80 mm. of mercury.

There was a loud systolic murmur in the pulmonic area as well as a blowing diastolic murmur in the same zone. Several pulsating intercostal vessels could be palpated over the posterior chest wall.

The radial pulses were full and forceful, while the pulsations of the abdominal aorta, femoral, posterior tibial, and dorsalis pedis arteries were markedly diminished.

Fluoroscopic examination and x-ray films of the chest showed an aortic knob, slightly smaller than usual, with no mediastinal enlargement. The descending portion of the aorta could not be well visualized. Motion of the heart and diaphragm were normal. The lung fields were clear and notching of the inferior rib margins was noted.

The electrocardiogram gave no evidence of myocardial disease. The blood counts and urinalyses showed no abnormalities. The blood nonprotein nitrogen and total serum proteins were normal and the phenolsulfonthalein test of kidney function showed 80 per cent excretion in ninety minutes.

This patient was operated upon on Dec. 21, 1946. A left posterolateral approach was employed and a segment of the fourth rib was removed. The intercostal arteries and veins were markedly dilated and at the insertion of the ligamentum arteriosum into the aortic wall a definite aortic constriction was present, below which no aortic pulsations were palpable (Fig. 1, *A*). The process of freeing-up the aorta was troublesome because of venous bleeding from a rich plexus of dilated vessels. The ligamentum arteriosum was divided and a 3 cm. segment of aorta containing the coarctation was excised between clamps (Fig. 1, *B*). The lumen at the coarcted level was but 4.5 mm. in diameter and a soft organizing thrombus was present at this point.

When an attempt was made to suture the aortic ends together, it was felt that the tension on the sutures was excessive and that it would be difficult and hazardous to try to complete the anastomosis. The only alternative was to utilize the left subclavian artery which was dilated to the same diameter as the aorta. Accordingly, the proximal aortic stump was closed and the left subclavian was freed up and divided between clamps at a point 4 cm. from the aortic arch (Fig. 1-*C*). The distal subclavian stump was sutured and the proximal subclavian stump was rotated caudad, Fig. 1-*D* and was anastomosed to the distal aortic stump using an everting suture of 3-0 Deknatel silk. (Fig. 1, *E* and *F*.)

Total operating time was five hours. Postoperatively, the patient's condition remained critical. Pulses in the legs were felt on only one occasion about eighteen hours after operation. Blood pressure in the legs could not be recorded. The radial pulse rate averaged 130 per minute and blood pressure readings in the arms remained high, averaging 200 mm. of mercury systolic and 80 mm. of mercury diastolic.

The patient died twenty-six hours after completion of the operation. At autopsy, he was found to have edema of the right lung and atelectasis of the left lung. The anastomotic site was partly obstructed by a thrombus. The degree of coronary artery sclerosis which was present in this young patient was quite remarkable.

Long intestinal clamps were used to occlude the vessel ends while this anastomosis was being completed, and it was quite difficult to keep the cut

ends of the vessels absolutely immobile in accurate end-to-end approximation. The result was that the fine sutures being used broke with annoying frequency. An attempt was made, therefore, to design a clamp which would firmly grasp the aortic wall between parallel bars without crushing, and which would permit the vessel ends to be accurately approximated to each other and immobilized.

Such clamps were designed and the original model was made through the kindness of Dr. J. Earle Thomas and Mr. William Goebbels, Professor of Physiology and Instrument Maker, respectively, at the Jefferson Medical College in Philadelphia.* This instrument is shown in Figs. 2 and 3.

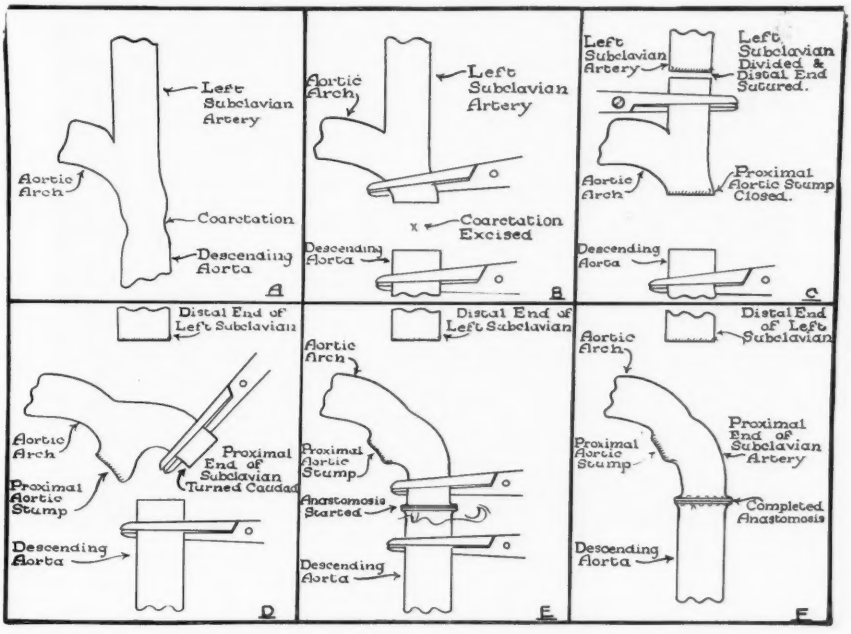


Fig. 1.—Diagrammatic representation of operative steps followed in the patient reported.

It consists of four parts: two clamps having parallel jaws 5 cm. long, the pressure on the jaws being accurately adjustable by means of a knurled nut; an octagonal rod 8 cm. long having a ball on either end, and threads on its four "corner" surfaces to receive a knurled nut. This rod can be inserted into square holes in each clamp so that it holds them in constant alignment with each other and permits them to be moved to or from each other. A handle, 18 cm. long can be attached by a ball-and-socket joint to either end of the bar. The handle can be moved through a wide range with respect to the bar and locked in any position desired.

*These clamps are now being supplied by the George P. Pilling & Sons Company, 3451 Walnut Street, Philadelphia, Pa.

This instrument has been employed experimentally on fifteen occasions and has proved satisfactory. When in use, the clamps can be removed from the bar or left on it and placed transversely across the aorta one above and one below the segment to be resected. The jaws can be closed to any desired pressure by turning the knurled nut, and the aorta can be thus occluded. It is important when applying these clamps to place the aorta in each clamp equidistant from the tips of the clamp so that when the clamps are joined by the bar and approximated, the aortic ends will be in accurate end-to-end apposition.

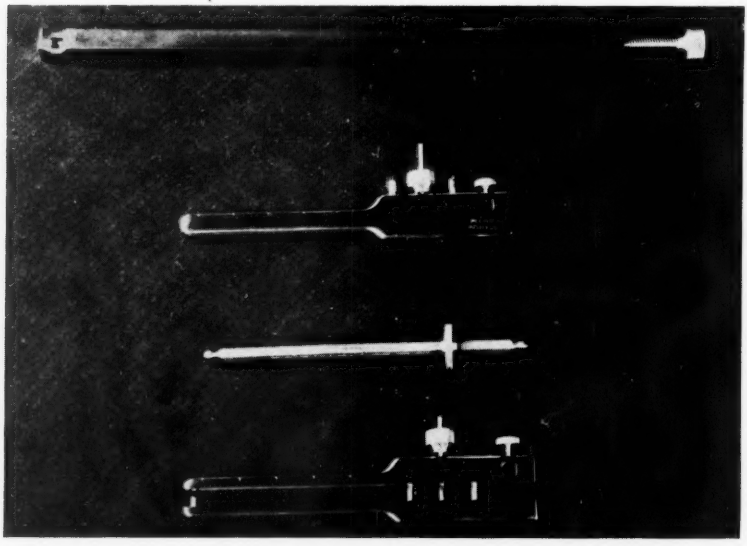


Fig. 2.—Photograph of the clamp disassembled to show its parts.

The desired segment of aorta can then be excised, leaving a free cuff about 8 to 10 mm. long on each side between the cut ends of the aorta and the clamps. (Fig. 4-*A*.)

If the clamps are removed from the bar before application, they can be joined by the bar after the aortic segment has been excised. The left hand clamp is locked on the bar by means of the small set screw, while the right hand clamp is left unlocked on the bar. Using the knurled nut on the bar, the right hand clamp can be moved toward its fellow (if desired, this relationship can be reversed) thus approximating the aortic ends. Both set screws are then tightened and the ends of the vessel are accurately secured in a satisfactory position for exact suturing. (Fig. 4, *B*.)

A running horizontal mattress suture of 4-0 braided silk has been used in the experiments done to date. This everts the aortic ends and allows for intima-to-intima healing. This suture is started on the upper surface of the

aorta at a point near the edge of the vessel which is farther from the operator. It is carried around the far edge of the aorta (Fig. 4, *B*). The clamp can then be rotated away from the operator (Fig. 4, *C*) and the suture line continued on the under surface of the aorta. When the nearer edge is reached, the clamp can be rotated again toward the operator and the suturing completed on the upper surface. (Fig. 4, *D*.)

Fig. 3.—Photograph of the assembled clamp.

The distal clamp can then be slowly released but left loosely astride the aorta should it be needed again. The proximal clamp can then be released very slowly over a period of several minutes and also left loosely across the aorta.

Should leaks occur, they can usually be controlled by steady pressure with a gauze sponge. If this is not sufficient, both clamps can be again tightened and an extra suture inserted.

The running horizontal mattress type of stitch is favored because it everts the edges nicely and causes much less strangulation of tissue than closely placed interrupted horizontal mattress sutures.

To date this clamp has been used experimentally on fifteen dogs. In each animal the aorta was approached through the left chest and segments of aorta varying in length from 4 to 12 mm. were excised.

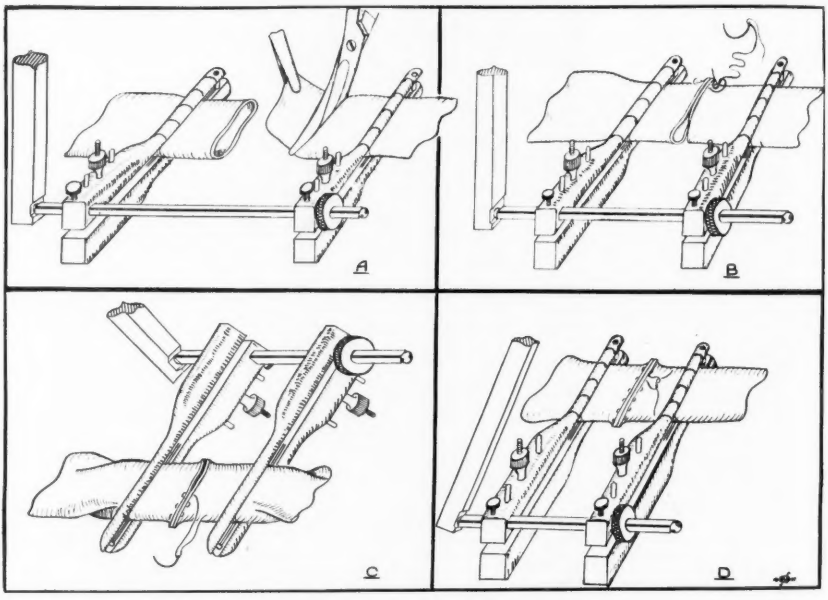


Fig. 4.—Diagrams of technical steps followed in the experimental resection and suture of the aorta. *A.* The clamps mounted on the horizontal bar have occluded the aorta and a segment is excised from between them. Note cuffs of aorta allowed to remain. *B.* The clamps have been moved together on the horizontal bar so that the aortic ends are apposed. A continuous horizontal mattress suture has been started on the "upper" surface and is being run around the edge farther from the operator. *C.* The clamp and aorta have been rotated away from the operator and the suture line is continued on the under surface. *D.* The clamp and aorta have been rotated back toward the operator and the suturing completed.

In the first ten dogs operated upon, sodium pentobarbital anesthesia was used in dosage of 30 to 40 mg. per kilogram of body weight. In each animal, pulmonary ventilation was carried out under positive pressure with atmospheric air administered by means of endotracheal tube and a motor-driven interrupter. In this group there was one survival and nine deaths. Three dogs died at the time of operation, one from anesthesia, and two from hemorrhage as a result of constant tearing of the aortic wall. Both dogs having friable aortic walls were older animals, and in addition to the sutures tearing through the coats, the layers of the aortic walls separated readily from each other. This raises the question as to the ability of an older aorta to retain sutures. Many observations on many human patients will have to be made before such a question can be answered, but it is of importance with relation to the optimum time for

resecting an aortic coarctation. Speaking from the purely technical standpoint of suturing, it may be preferable to resect aortic coarctation early in the life of the patient before the aortic wall proximal to the coarctation has become physiologically old.

In this same group of ten dogs, two died within twenty-four hours with paralysis of the hind legs, bowel, and bladder; two died at four and six days after operation, respectively, from hemorrhage; one died on the fifth day of infection; and one died on the sixth day with paralysis of the rear legs, bowel, and bladder.

Thus four out of seven dogs surviving the operation, per se, had paralysis of the rear legs, bowel, and bladder. These dogs had large bowel evacuations while the aortic clamp was in place as well as after its removal.

It is felt by many investigators in this type of work that these paralyses are the result of ischemia of the spinal cord. The length of time during which the aorta can be clamped without developing a paralysis of hind legs in a dog has been variously estimated by many observers, but is usually considered to be about fifteen minutes. There is, however, an apparent variation in this period of occlusion, for in dogs with aortas occluded thirty to forty-eight minutes, paralysis has not occurred.

Various measures have been used in attempts to overcome this complication. Gross and Hufnagel,[7] in their experimental work, cooled the lower part of their animals in order to lessen the severity of this complication. We have considered the use of some by-passing operation similar to those used by Carrel[8] in 1910, and also the use of transfusions into the distal aorta while the aortic clamps are on. How extensively this type of procedure should be investigated is a matter of conjecture, because conditions in these dogs are far different from those in human patients with coarctation where an extensive collateral circulation already exists. However, we did feel it worth while to investigate the possibility of the use of high concentrations of oxygen to see whether it would have a beneficial effect in reducing these neurologic complications. Accordingly, in the next five dogs operated upon, the same anesthesia and the same technique was used, but, in addition, oxygen at the rate of 4 L. per minute mixed with room air was administered through the positive pressure respiratory apparatus. It was felt if the oxygen saturation of the blood could be raised to its maximum point before the aortic clamps were applied, then while the clamps were in place and the aortic circulation completely obstructed, the tissues distal to this site would have a somewhat larger reserve of oxygen to help to tide them over the period of occlusion.

Three dogs breathing room air after administration of sodium pentobarbital anesthesia were found to have an average oxygen saturation in their venous blood of 74.4 per cent. After the aortas had been occluded for 20, 25 and 25 minutes, respectively, the average oxygen saturation in femoral vein blood was 11.6 per cent, while oxygen saturations in venous blood from the forelegs averaged 80.1 per cent. The hemoglobin values in these dogs varied from 8.7 to 12.1 Gm. per cent.

Two other dogs breathing room air after administration of sodium pentobarbital anesthesia had average oxygen saturations in their venous blood of 82.1 per cent. These two dogs were then given oxygen at a rate of four liters per minute mixed with room air via an endotracheal tube and the oxygen saturation of venous blood rose to an average of 98.4 per cent. Oxygen plus room air was continued while the aorta was occluded. After periods of aortic occlusion of twenty-three and twenty-eight minutes, respectively, the oxygen saturation of femoral vein blood averaged 16.6 per cent, while the oxygen saturation of venous blood from the foreleg averaged 101.0 per cent. The respective hemoglobin levels in these dogs were 12.6 and 13.5 gm. per cent.

Thus, by administering high concentrations of oxygen to these animals, the oxygen saturation of femoral vein blood after twenty-five to thirty minutes of aortic occlusion was increased from 11.6 per cent to 16.6 per cent. Further, none of the dogs receiving oxygen had any paralysis of their hind quarters, and the occurrence of profuse bowel evacuations was greatly reduced. Of five dogs receiving supplementary oxygen, two died eight and eleven days, after operation respectively, as a result of hemorrhage from the site of anastomosis. The other three are living and well 56, 75, and 76 days postoperatively.

There are certain details of this clamp which can be improved upon, but so far in its experimental use the basic principles of its design have proved useful. As further experience accumulates experimentally and clinically, it will doubtless be modified and it may be advantageous to make a somewhat similar instrument in different sizes for use in patients of varying sizes.

We are deeply grateful to Dr. James W. Dickey, Jr., and Dr. Louis Shaffner for their very generous assistance in operating upon these dogs and carrying out the determinations of blood oxygen.

REFERENCES

1. Hines, E. A., and Christensen, N. A.: Diagnosis and Prognosis in Cases of Coarctation of the Aorta, Proc. Staff Meet. Mayo Clin. **22**: 121, 1947.
2. Crafoord, C., and Nylin, G.: Congenital Coarctation of the Aorta and Its Surgical Treatment, J. THORACIC SURG. **14**: 347, 1945.
3. Gross, R. E.: Surgical Correction for Coarctation of the Aorta, Surgery **18**: 673, 1945.
4. Clagett, O. T.: Coarctation of the Aorta: Surgical Aspects, Proc. Staff Meet. Mayo Clin. **22**: 131, 1947.
5. Gross, R. E.: Surgical Treatment for Coarctation of the Aorta. Modern Concepts of Cardiovascular Disease, Feb., 1947.
6. Blalock, A., and Park, E. A.: The Surgical Treatment of Experimental Coarctation (Atresia) of the Aorta, Ann. Surg. **119**: 445, 1944.
7. Gross, R. E., and Hufnagel, C. A.: Coarctation of the Aorta, New England J. Med. **233**: 287, 1945.
8. Carrel, A.: On the Experimental Surgery of the Thoracic Aorta and the Heart, Ann. Surg. **52**: 83, 1910.

DISCUSSION

DR. HOWARD BRADSHAW, Winston-Salem, N. C.—We tried the clamps Dr. Gross had suggested, and we found a great deal of difficulty in holding the aortic ends together. With each pulsation, of course, they gave a bit, and sutures pulled out, particularly if, as you have noticed in the movie, the lower end of the aorta is diseased. Stitches pull out easily in such a blood vessel.

We have used the clamp in dogs, and it works well with them. We have not had occasion yet to use it in patients.

Any of you who are interested in this clamp can see it some time during the meeting. It was made by the Department of Physiology, Jefferson Medical College, Pa. I am sure there are many wrinkles that can be improved upon, and we would appreciate suggestions.

We wanted to get a clamp that would not slip and would not be in our way too much. It is a very simple idea, and we do not deserve any credit for originality. We put that handle on so that it is possible to use it at most any angle desired.

This does hold the severed ends in approximation without any doubt. The clamps can be adjusted tightly or loosely, and so far it has seemed in normal aortas, at least, in dogs, that it is quite an adequate instrument for an end-to-end anastomosis of the aorta.

In the movie you noticed there was not much left of the upper stump of the aorta, and I think it is wise to have a better clamp than just an ordinary stomach or intestinal clamp with someone holding it. One can rotate this clamp either way, and put the posterior sutures first.

There is no doubt about being able to secure an accurate approximation of the aortic ends. We have operated upon some fifteen dogs with it, and the anastomosis has been uniformly satisfactory. We have found that—with our very limited experience—the time that one can clamp off the aorta is very variable, and we think that an oxygen-rich atmosphere helps a great deal in preventing hind leg paralyses.

DR. STUART W. HARRINGTON, Rochester, Minn.—At the present time this procedure is essentially an operation of necessity rather than one of choice. For that reason, I believe it is important to evaluate it as to whether it is a justifiable palliative operation to substitute for resection of the stenosed area and end-to-end anastomosis.

Dr. Clagett has done several of these operations, and I am sorry that he is not here to report his experience with this procedure. He has given me a summary of the cases he has done, to present to you, which I will read.

"Our experience with the anastomosis of the subclavian artery to the thoracic aorta in the treatment of coarctation consists of five cases at the present time. In each case the patient was explored with the hope that it would be possible to do the classical type of operation with excision of the stenosed area and an end-to-end anastomosis. In these cases, either because of the location or the extent of the stricture, it was not feasible to carry out this technique, and in each the subclavian artery was used. In four cases the stenosed area was ligated, the aorta was sectioned just distal to the stricture, the subclavian artery was sectioned, the distal end ligated, the proximal end rotated over, and an end-to-end anastomosis to the thoracic aorta achieved. In the fifth case the stenosis of the aorta was not complete. There was a thrill indicating some blood going through the strictured area. In that case the end of the subclavian artery was anastomosed to the side of the thoracic aorta. The patients were 34, 31, 28, 21, and 18 years of age. All had rather severe hypertension in the upper body, with blood pressures ranging from 185 to 230, and in all the blood pressures in the lower extremities were low or unobtainable. The most predominant symptoms were headache, dizziness, and weakness. Two patients complained of severe weakness in the legs after moderate exercise. Three of the patients had been referred to the clinic for sympathectomy because of a diagnosis of hypertension; in the other two a diagnosis of coarctation had been made elsewhere previously.

"The postoperative course of three of the patients was uneventful, and they were dismissed from the hospital in good condition. The fourth patient had a little persistent fever and died on the thirty-second postoperative day from hemorrhage due to separation of the posterior wall of the anastomosis. The last patient is still in the hospital. Her postoperative course has been satisfactory. All the patients but one obtained a satisfactory decrease in their arm blood pressures postoperatively, and simultaneous increased femoral blood pressures. The patient who did not obtain such a satisfactory result is the one in whom the end-to-side rather than the end-to-end anastomosis was performed. The first patient we operated on had a blood pressure of 230/140 before operation, and when seen recently, six months later, his arm pressure was 152/80 and his leg pressure, 154/86.

"This group of patients is older than is desirable for surgery, since in every case there was a rather severe degree of sclerosis of the vessels. It certainly would be preferable

to operate on younger individuals; however, we have not felt that these patients should be denied operation. There has been no difficulty with the left arm resulting from ligation of the subclavian artery. The arm is a little cooler and radial pulsations cannot be felt, sometimes, for days or even weeks after operation, but there has been no weakness or disability and collateral circulation appears to be entirely adequate.

"There are obvious disadvantages to the use of the subclavian artery for the anastomosis rather than making an end-to-end anastomosis of the aorta after resection of the stricture. The use of the subclavian for anastomosis to the aorta does not increase the vascular bed through which blood is evacuated from the heart, but the resistance is materially decreased and allows a more normal adjustment in blood pressure in the upper and lower portions of the body. The advantages in the use of the subclavian are that an anastomosis can be performed without tension on the anastomosis, and that it is possible to perform the operation in a patient in whom the stricture is too long to permit an end-to-end anastomosis.

"It is much too early to evaluate the end results of these patients. They all have had a rather severe degree of arteriosclerosis, and we cannot anticipate these blood vessels will return to a normal condition. However, the decrease in blood pressure to the upper body and the improvement in the blood supply to the lower body should give the patient a great deal of protection against cardiac failure and cerebral accidents. From our experience so far, we are encouraged and believe the procedure has a very useful place in the treatment of those patients in whom it is impossible to establish an end-to-end anastomosis of the aorta."

DR. JULIAN JOHNSON, Philadelphia, Pa.—I would like to show some slides on a couple of these patients that we have had occasion to operate upon in the last few months.

(Slide.) These patients both had the so-called infantile type of coarctation, in that the coarctation extended over a long area. By actual measurement, the area that had to be resected here was in the neighborhood of 4 or 5 cm. Obviously, it would be impossible in this patient to do an end-to-end anastomosis.

In this slide the head is upward, the descending aorta down below. The vessel going upward is the large subclavian artery. We were not aware that anyone had used the subclavian before, but it was such a logical procedure that we went ahead with it.

(Slide.) This was a 13-year-old boy. As you bring it down, you see the difference in the size of the subclavian and the descending aorta, and this boy, where he had a previous blood pressure in the neighborhood of 170 over 110 to 120, has dropped down now to a blood pressure of 100 over 70, and has good, bounding pulses in his leg.

(Slide.) That boy has had an excellent result as far as we can see. Now, three months later, he has as good a result as he could have gotten by any means.

(Slide.) On the other hand, here is an 18-year-old boy. The first one had a complete obliteration of his aorta. This was not complete. Here you cannot see the subclavian up above, but we thought that the subclavian artery was larger than the little stump of descending aorta, and so elected to do the same procedure.

(Slide.) Again, we had to resect about 4 cm. of the aorta. As you see, in this patient we thought that there was a greater difference in the size between the subclavian and the descending aorta. This boy had an excellent bounding pulse in his feet, immediately after operation. The next day, however, it had diminished considerably, although we could still feel it. We thought that perhaps he must have had some thrombosis going on at the site. It gradually came around again, and within a period of a month after operation had improved considerably. His blood pressure, however, has not dropped to normal. It was in the neighborhood of 200 before operation. It has come down to 170, and not returned to normal. We presume that whereas there was some flow through the aorta at the time, we are not sure we increased his blood flow, and have not increased it sufficiently to return him to a normal blood pressure.

DR. JOSEPH W. GALE, Madison, Wis.—The first case which we did about three months ago turned out beautifully. We did a good end-to-end anastomosis. The coarctation was about a centimeter below the base of the subclavian. We placed a homemade clamp above it which worked very well.

The individual had systolic pressure of 200 to 220 over 100 and 110 previous to the operation. Immediately after the operation it fell to normal and has remained there.

In the second case, the coarctation was so close to the subclavian that we could not place a clamp above it, so that necessitated utilizing the left subclavian. There was quite a difference in the size of the distal aorta and the subclavian, so we had to cut the latter on a bias. We performed a fair anastomosis. This 18-year-old patient had a blood pressure of 170 over 100, following the operation it fell to 120 over 70. This pressure was maintained for three and one-half days. Since then it has risen, until now, fifteen days after operation, it varies between 180 and 210.

I think there is something wrong. I don't know what it is, but suspect that there is a thrombosis at the site of the anastomosis.

I would like to ask some of you gentlemen who have had more experience what the chance of thrombosis is in these cases.

We did a number of these operations on dogs, previously, and it probably took us too long to perform the operation, since they all developed paralysis of the lower extremities, and every one developed a thrombosis. Now if a thrombosis has occurred in our second patient, have we done him any harm, or will there be some recanalization and improvement later? Right now we are rather discouraged about the outcome of this case.

DR. HOWARD T. BARKLEY, Houston, Texas.—I just wish to add one case, interesting from the standpoint of pathology.

This was a patient 40 years of age, completely incapacitated by the most severe cerebral hypertension. At operation we found a greatly thickened aortic wall, and at the site of the resection of the coarctated area, a thickened atheromatous plaque was encountered on the medial wall of the proximal cut end of the aorta; therefore, we had to resect a triangular segment out of the aortic wall, just distal to the subclavian artery, to remove this plaque. Anastomosis was accomplished.

This patient died forty-eight hours postoperatively, with sudden pain and shock. We supposed that our anastomosis had given away and he had bled, but on post-mortem examination the anastomosis was intact. It was found that the Crafoord clamps had been placed across other atheromatous plaques above and below the point of anastomosis and had fractured the intima over these plaques. A mural thrombus had formed, which was attached at the site of the fractured intima above and below the anastomosis but was not attached to the suture line. The lumen of the aorta was not occluded, and we were unable to determine the cause of death. There was no coronary thrombosis.

DR. H. BRODIE STEPHENS, San Francisco, Calif.—We have employed retrograde injections of 35 per cent Diodrast solution to demonstrate coarctation of the aorta. We have injected 25 c.c. of the 35 per cent solution into the left common carotid artery of several children around 4 years of age where the diagnosis of coarctation was questionable by clinical examination. The left common carotid artery is exposed in the neck and a No. 18 gauge needle inserted retrograde into this blood vessel. The diodrast solution is injected as rapidly as possible while roentgen films (left lateral oblique) of the chest are taken in rapid succession during the injection of the diodrast solution. This procedure is carried out under general anesthesia. An excellent demonstration of the aortic neck, the thoracic aorta and the abdominal aorta is obtained by this technique. We have not obtained any ill effects from retrograde injections carried out in the manner just described.

DR. LOUIS R. DAVIDSON, New York, N. Y.—In discussing this interesting presentation of Dr. Bradshaw I should like to make two observations which are based upon the experiences gathered by Dr. Jere Lord and myself. At Post-Graduate Hospital in New York City we have operated upon different types of congenital hearts; at Cornell Medical College we have performed our animal work. In consequence of our experiences we have come to the conclusion, as most of you have, that these strictures of the aorta must be considered from two viewpoints—extent and location.

It is obvious that a long stenosis of the aorta precludes operation of the orthodox type since the ends of the aorta cannot be approximated and sutured. At times, even in the

so-called average case, it is difficult to have the sutures hold in the vascular wall. The location of the coarctation also creates problems. In one of our cases, lantern slides of which will be shown in a moment, we were quite apprehensive throughout the entire operation by virtue of the fact that we had but 3 mm. of aortic cuff projecting from the proximal clamp. This clamp just avoided the left subclavian artery. Because of this experience we have performed the following operation which we hope will obviate this difficulty, thereby making the procedure less hazardous. I may say parenthetically that this operation has been performed only in the animal laboratory and as yet not upon a human.

The aorta and the left subclavian artery along with the intercostal arteries are mobilized in the customary fashion. This holds true for the ligamentum arteriosum. At the start, like most others, we were very cautious in our approach to the left vagus and the left recurrent laryngeal nerves. We have lost our fearfulness and have found that there is no apparent disturbance following careful liberal mobilization of these neural structures. When the vessels and the nerves have been adequately prepared and particularly the aorta freed posteriorly a Blakemore-Crump clamp is placed upon the aorta between the left carotid and the left subclavian arteries. Over the distal aorta a Bainbridge clamp is placed and on the left subclavian artery a bulldog clamp is applied. This permits sectioning of the aorta flush with the take-off of the left subclavian artery, removing the coarctation distal to the section. Then, without rotating the vascular structures, an anastomosis between the distal aorta and the aortic arch is made. We propose this operation for those cases of coarctation of the aorta where the problem of applying the proximal clamp arises in consequence of the high stenosis. Dr. Lord and I believe this procedure has certain merits:

A. High strictures can be readily handled without sacrificing a vessel.

B. Rotation of the aorta is unnecessary.

C. Alarming hemorrhages can be avoided.

Just one other matter: Dr. Maier, I believe, in his presentation, by indirection implied that all cases should have an angiocardiogram. We subscribe to that statement quite definitely.

(Slide.) This angiocardiogram was made for us postoperatively by Dr. Henry K. Taylor. You note that the proximal aorta is fully visualized.

(Slide.) Here one sees the distal aorta well outlined.

(Slide.) In this third slide we obtained information which, if known earlier, would probably have precluded surgery. The left subclavian artery is not visualized and probably is absent. The right subclavian artery arises from the left side of the arch of the aorta. One notes a marked collateral circulation with a very markedly enlarged right internal mammary artery. In this case the coarctation was just distal to the left common carotid artery. Here, in desperation, because of the location of the constriction, the end of the left third intercostal artery was anastomosed to the side of the carotid artery. This patient, 10 years of age, obviously has failed to show any improvement. Fortunately his condition has not been made worse.

DR. JAMES F. O'NEILL, Winston-Salem, N. C.—Because we stopped the movie before it had fully run through, I don't think that we could appreciate the severity of the coarctation he had. Looking at it from the outside of the aorta, it did not seem to be very great, but after a specimen was excised, the internal lumen of this coarctated area was only about 3 mm.

Second, at autopsy this patient showed marked coronary sclerosis, and a great deal of sclerosis in the aorta proximal to the coarctation. This may be a physiological aging process, but we are not certain of that.

AORTIC-PULMONARY ANASTOMOSIS FOR PULMONARY STENOSIS

WILLIS J. POTTS, M.D. (BY INVITATION)
CHICAGO, ILL.

CYANOSIS due to certain types of congenital heart disease can now be relieved surgically in a fair percentage of cases. It must be demonstrated that the patient has an insufficient flow of blood to the lungs as manifested by cyanosis, increased red blood cell count, increased hemoglobin, decreased oxygen saturation of arterial blood and a markedly decreased tolerance for exercise for the condition to be suitable for surgery. In patients with typical pulmonary stenosis as part of the syndrome of the tetralogy of Fallot, x-ray and fluoroscopic examination should reveal: a fairly typical boot-shaped heart with a concavity in the region of the pulmonary conus on the left, a "pulmonary window" in the right anterior oblique and left anterior oblique views, and absence of pulsations in the hilar regions of the lungs. A barium swallow will usually reveal whether the aortic arch curves to the left or to the right. A concavity in the barium on the left side of the esophagus indicates a left aortic arch and a concavity on the right side indicates a right aortic arch. The electrocardiograph in these patients must show a deviation of the axis to the right. In children with tricuspid atresia the heart is likely to be rather globular and slightly flattened on the right in the region of the right ventricle. The electrocardiograph in such patients shows a deviation of the axis to the left. A cyanotic child with a large heart or a prominent pulmonary conus, with increased hilar markings or visible pulsations in the lung fields on fluoroscopic examination does not fall in the operable group.

Operative success will depend upon an accurate diagnosis of the type of congenital heart disease and a careful selection from this group of those who may be benefited by surgery. The diagnostic work at the Children's Memorial Hospital is done by Dr. Stanley Gibson. Because of publicity it is quite natural for each mother of a cyanotic child to anticipate that her unfortunate youngster can be successfully operated upon. This is far from the truth. In our experience approximately one-half of the children with varying degrees of cyanosis due to cardiac abnormalities have conditions which are unsuitable for operation. Even so, we have operated upon a number of children in whom the diagnosis was questionable but whose condition was such that exploration was indicated since surgery offered the only chance for survival.

Following the outstanding work of Blalock and Taussig[1] in this field of surgery, interest in these hitherto hopeless cyanotic children has revived. In a large series of patients they have been successful in transforming anoxic children from helpless to happy and fairly normal individuals. To increase the flow of blood to the lungs, Blalock anastomoses one of the large systemic vessels

From the Children's Memorial Hospital and from the department of experimental surgery, Northwestern University Medical School, Chicago, Ill.

Read at the Twenty-seventh Annual Meeting of The American Association for Thoracic Surgery, St. Louis, Mo., May 28, 29 and 30, 1947.

to a pulmonary artery. Use of the subclavian has resulted in no permanent damage to the arm but use of the innominate or carotid arteries has led, in a certain percentage of cases, to transient or permanent damage to the brain. Because of this danger, it occurred to me that it might be possible to establish a ductus arteriosus between the aorta and a pulmonary artery if by some means a fair portion of the blood could be allowed to flow through the aorta while the anastomosis was being made. In November, 1946, a new procedure for the relief of cyanosis due to pulmonary stenosis was published with Drs. Smith and Gibson.[2] The principle worked out in the experimental laboratories at Northwestern University Medical School involved in the use of a clamp which encircles the aorta and pinches off a small lip of it to which a pulmonary artery can be anastomosed. At the same time the clamp allows plenty of blood to continue to flow through the partially constricted aorta (Fig. 1). Although the technique has remained substantially the same as previously described, a few details and their significance are worthy of note.

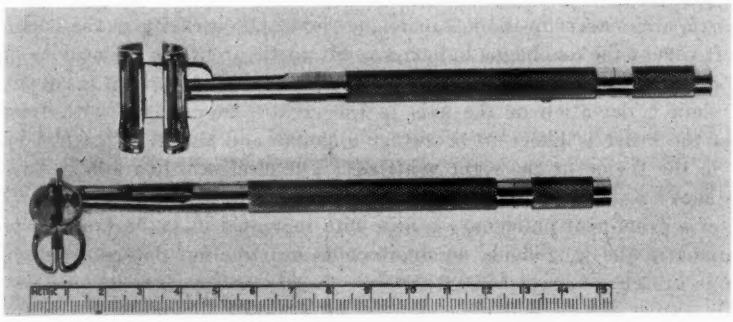


Fig. 1.—Aortic clamp.

I cannot emphasize too strongly the importance of expert anesthesia. All patients operated upon in this series were anesthetized by Dr. William O. McQuiston. Cyclopropane has been the anesthetic agent of choice because of the high concentration of oxygen that can be given with it. Ordinarily these patients become less cyanotic as soon as the anesthesia is begun. The actual technique of anastomosis will be tremendously aided by the anesthetist who is able to stop reflex respirations and "breathe for the patient" by rapid intermittent pressure on the anesthetic bag. This maneuver stops all movements of the diaphragm and allows the aorta and pulmonary artery to lie absolutely motionless. Curare in small amounts has been of great assistance in controlling respiration.

It will be noted from the illustration (Fig. 2, *E*) that the posterior row of sutures apposes the aorta to the pulmonary artery, adventitia to adventitia. There was considerable skepticism in our minds about the propriety of this type of suture because it has long been wisely preached that in blood vessel surgery intima should be sewed to intima. In this operation it is technically

impossible to suture the posterior row intima-to-intima. Microscopic study of sections cut from the walls of the anastomotic channels produced in dogs indicates that it is unnecessary in aortic-pulmonary anastomosis to appose the

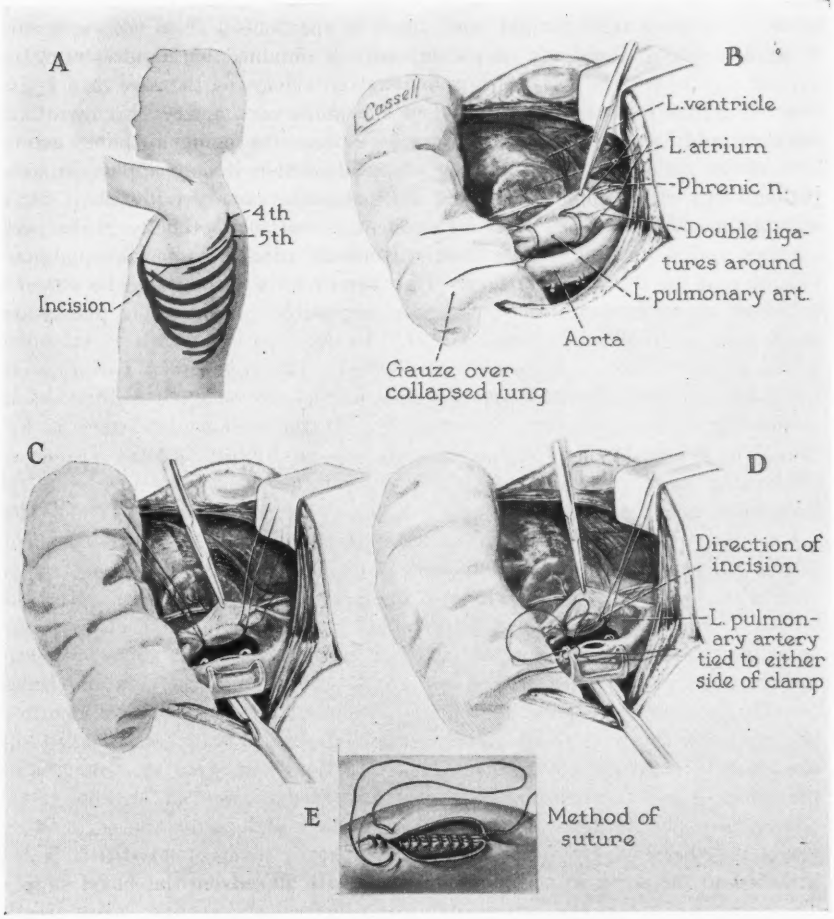


Fig. 2.—Technique of aortic-pulmonary anastomosis.

the vessels intima-to-intima. The difference in blood pressure in the aorta and the pulmonary artery is so great that the constant strong flow of blood through the new channel will keep it open. Within a short time a surface clot is formed over the rough edges and the exposed silk. This layer of fibrin in turn is rapidly covered with endothelium. While I do not wish to say that intima-to-intima suture in general is not preferable, I do not believe that it makes any difference in this operation where the differential in pressures is so

great (Fig. 3). In all of the children who have survived surgery, there has been a persistent soft murmur in both systole and diastole indicating that blood is continually flowing through the newly formed channel.

The patient with a left aortic arch is laid on the operating table on the right side and the chest is opened by an incision through the left fourth interspace. A patient with a right aortic arch is approached from the right side through a similar incision. Ample exposure is obtained without resecting or cutting any ribs. The left or right pulmonary artery, as the case may be, is freed from the hilus of the lung. The left pulmonary artery is longer than the right and does not divide into branches as near the main pulmonary artery as does the right. At the beginning of the dissection it may appear in some patients with right aortic arches that the pulmonary artery is too short for a suitable anastomosis to the aorta. Persistent dissection proximally to the pericardium and meticulous freeing of the adventitial adhesions allow adequate mobilization of the pulmonary artery. Thus far we have encountered no instance in which an anastomosis was technically impossible, provided the pulmonary artery was of sufficiently large caliber. In four patients with right aortic arches successful anastomoses were performed. The tremendous variations in the caliber of the pulmonary arteries from normal size to a few millimeters in diameter are unpredictable preoperatively. If the pulmonary artery is less than 3 or 4 mm. in diameter, anastomosis will be difficult and the wisdom of attempting it will be questionable. The small tortuous veins overlying the pulmonary artery and aorta in practically all cases must be dissected off. This not infrequently occasions slight oozing of dark blood which obscures the field. We have discontinued the use of sponges in the chest because they cause trauma and fail to pick up the blood caught in the meshes of loose adventitia. Instead, with a 50 c.c. bulb syringe, physiological solution of sodium chloride is forced in a stream over the operative site and is removed by suction. This procedure not only washes away the excess blood but brightens the structures and makes possible their identification. After the pulmonary artery has been identified and well freed from its bed, doubly encircling ligatures of fairly heavy silk are placed around the proximal and distal portions and about any intervening branches. These ligatures are well oiled to prevent them from sticking to the adventitia. The ligatures are left loose and laid aside while the aorta is exposed and dissected from its bed. A fairly heavy layer of adventitia is left attached to the aorta so as not to interfere with its adventitial blood supply. The number of intercostal arteries requiring ligation and cutting varies greatly from one to twelve. The average is about five.

The medium-sized clamp previously described is used for most patients. The small clamp has been used on children below one year of age; the large clamp on one child 12 years of age. The anastomosis will be simpler technically if a fairly large lip of aorta is clamped off. Palpation of the aorta distal to the clamp will reveal a definite thrill if sufficient blood is going through after the clamp has been tightened.

The incision in the aorta is made from 5/16 of an inch (8 mm.) to 4/16 of an inch (6.3 mm.). In each case the opening is carefully measured with a

caliper. In the last six patients we have made the incision in the aorta 4/16 of an inch (6.3 mm.). Since the immediate postoperative results have been just as good as in those patients who have a larger opening, we are naturally inclined for the moment to continue with the smaller incision. The anastomotic channel should be large enough to relieve cyanosis but not large enough to embarrass the heart. What the optimum diameter of the newly formed ductus arteriosus should be can only be determined by the study of patients postoperatively over a period of years. In one child, extremely cyanotic, with a red blood cell count above 10,000,000 the incision in the aorta was made 6/16 of an inch (9.5 mm.). Although the child has been completely relieved of all visible cyanosis the very loud "humming top" murmur makes us fearful that in the course of years she will have difficulty because of excessive strain on the heart. However, to date there has been only the usual moderate amount of cardiac enlargement seen in all patients postoperatively.

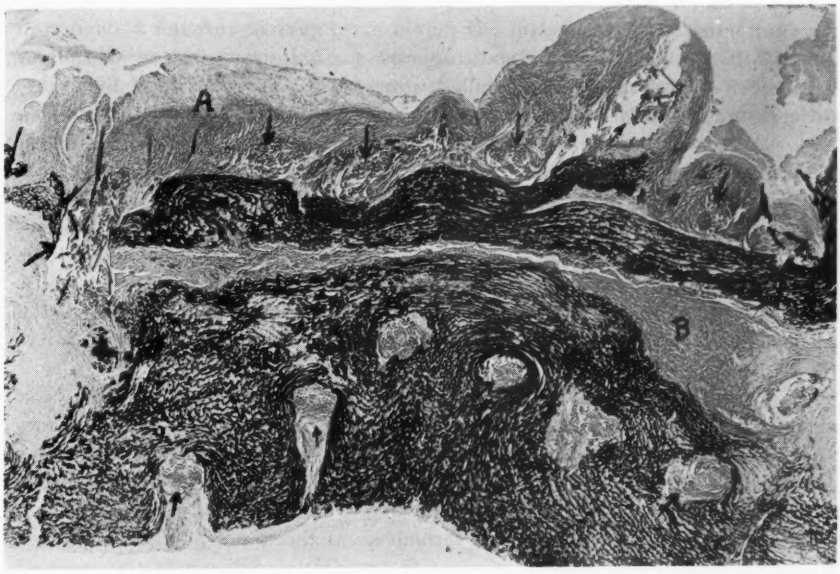


Fig. 3.—Microscopic section, Verhoff's stain; magnification ×60, taken from anastomotic channel 8 days after aortic-pulmonary anastomosis in a dog. The section is cut transversely from the posterior edge of the anastomotic channel where the vessels were sewed together adventitia to adventitia. *A*, Intima. *B*, Adventitia. The elastic tissue in the media is stained black. The upper layer is the pulmonary artery and the lower layer is the aorta. Note that the silk sutures (indicated by arrows) are buried and completely covered with intima.

A slight modification in tying the pulmonary artery to the clamp has been made and it simplifies the technique (Fig. 2, *D*). Instead of tying the pulmonary artery to the anterior lip of the clamp only, one end of the ligature is carried through the posterior lip of the clamp first and then through the anterior. The other end of the ligature is threaded through the same lips in the opposite direction. This brings the pulmonary artery to the aorta in

better position for anastomosis. The ligatures are drawn snug and tied while the assistant presses the pulmonary artery against the clamp with a right angle forceps. The ligatures not only shut off the blood supply but also hold the pulmonary artery in position for anastomosis.

The cut in the pulmonary artery is made a little smaller than the cut in the aorta because during suture it tends to stretch. The anastomosis is made with fine 5-0 Deknatel arterial silk on a No. 9 curved atraumatic needle employing a continuous over-and-over running suture. The method of sewing has not been changed. Upon release of the ligatures and clamp, there has been very little bleeding from the suture line. Except in one case in which there was brisk bleeding at the lower angle of the anastomosis, no extra sutures have been necessary. The clamp was reapplied and the bleeding stopped with one suture. Palpation of a thrill at the anastomotic channel gives comforting reassurance that blood is flowing through the new channel into the pulmonary artery.

Upon completion of the anastomosis, the lungs are carefully re-expanded by the anesthetist. A drainage tube is put in every patient through a small stab wound between the ribs in the sixth interspace. A Pezzar catheter with all of the tip cut off but the flange is pulled through the chest wall from the inside out and attached to a water seal as soon as the patient is returned to his room. The catheter is left in three days. The policy of putting a drainage tube in the chest in all patients is the result of a death due to tension pneumothorax.

When the operation has been completed and the dressings have been applied, the patient is slowly turned from the lateral to the supine position. Sudden shifts in position have on two occasions caused marked changes in the pulse and blood pressure due presumably to mediastinal shift. As soon as it is apparent that the respiration, pulse and blood pressure have stabilized, the patient is returned to his room and placed in oxygen for twenty-four to forty-eight hours.

To date, aortic-pulmonary anastomosis has been performed on twenty-six patients with pulmonary stenosis (tetralogy of Fallot) and on three patients with tricuspid atresia, a total of twenty-nine patients. There have been four deaths, or a mortality of 13.8 per cent (Table I). The causes of death were as follows: a girl, aged 12, died fifteen hours after operation. Post-mortem examination revealed fresh cerebral thrombosis of the upper portion of the left postcentral gyrus. A scar in the right frontoparietal region was the cause of hemiplegia during infancy. A 20-month-old child died eighteen hours after operation without regaining consciousness. Post-mortem examination revealed an area of softening in the left temporal region. From gross examination to date it is impossible to say whether this is an old or a fresh lesion. The child had had hemiplegia at the age of 15 months. The causes of death were anoxia, shock and cerebral thrombosis (?). The third death in a 4-year-old girl twelve hours after operation was due to tension pneumothorax. At operation an unsuspected tuberculosis was found in the left apex not demonstrable by x-ray examination. Presumably the lung was opened during the process of freeing it from the parietal pleura. Although the lung expanded normally at the

close of operation, at postmortem examination it was completely collapsed and the heart had been shifted to the right. In all these patients the anastomosis was intact. The fourth death in a 33-month-old boy occurred three days after operation. During the operation *before* the clamp was applied to the aorta the systolic blood pressure rose to 152 mm. Hg and the diastolic pressure to 120 mm. Hg. Application of the clamp caused a rise in the systolic blood pressure to 162 mm. Hg. Postoperatively he was extremely restless, breathed very heavily and complained of excessive thirst. A presumptive diagnosis of hemorrhage in the hypothalamus was made. Post-mortem examination unfortunately was denied.

Five operations on four patients proved to be exploratory only. One had an Eisenmenger complex not amenable to surgery; the others had such small pulmonary arteries that anastomosis was impossible. Two of the latter died. At post-mortem examination, atresia of the pulmonary artery was found in both cases. All of the blood to the lungs was carried by bronchial arteries arising from the arch of the aorta for the right lung and from the descending portion of the aorta for the left lung.

While the most suitable age for operation is probably between 3 and 7 years, we have performed anastomoses in thirteen children who were below 3 years of age (Table II). Successful anastomoses were performed on two children below one year of age; one four months and one 6 months.

TABLE I. MORTALITY AND RESULTS

Aortic-pulmonary anastomosis performed	
For pulmonary stenosis	
Tetralogy of Fallot	26
Tricuspid atresia	3
Total	29
Deaths	4
Mortality	13.8%
Those who survived surgery	
Markedly improved (cyanosis largely relieved. Patients living fairly normal lives.)	24
Improved (four-month-old child, cyanosis still present when crying.)	1
Total	25

TABLE II. AGES OF ALL PATIENTS OPERATED UPON

AGE	NO.
4 months to 1 yr.	2
1 to 2 yr.	8
2 to 3 yr.	3
3 to 6 yr.	10
6 to 9 yr.	5
9 to 12 yr.	5
Total	33

TABLE III. AVERAGES OF PATIENTS WHO SURVIVED ANASTOMOSIS

	PREOPERATIVE	2 WEEKS POSTOPERATIVE
R.b.c. count	7,930,000	5,620,000
Hemoglobin	20.87	15.6
Hematocrit	64.4	51.
Oxygen saturation of arterial blood	47.3	77.9

The red blood cell count dropped an average of over two million within two weeks after operation (Table III). Comparable drops in hemoglobin occurred and yet there was no visible jaundice and in the few patients examined there was no elevation of the icteric index. Oxygen saturation levels made on blood drawn from the femoral artery are likely to give false impressions because the children usually struggle while the blood is being drawn. A true basic reading is rarely obtained. Of far more importance to us is the exercise tolerance test. Those patients old enough to walk are led up and down a measured corridor in the hospital and the point at which they are forced to stop and sit down is noted both preoperatively and postoperatively. In five of the children the exercise tolerance was zero. In fact, two of these children could not be taken out of oxygen preoperatively. Strangely enough, all five survived surgery. The highest exercise tolerance was 486 yards before cyanosis became severe. The average child could walk 100 to 200 yards before assuming the squatting position which for some reason seems to give quickest relief.

Postoperative complications have not been troublesome. It seems that the child either survives the operation uneventfully or dies. Four patients postoperatively had bloody fluid and/or air in the chest which required one or two aspirations. Since the introduction of a drainage catheter in the chest at operation there has been no such trouble. One child had postoperative tracheobronchitis and was placed in a steam room for a few days. One patient, previously reported, a 6-year-old girl, extremely anoxic with an exercise tolerance of a few steps, remained very cyanotic throughout the operation. She did not arouse normally upon return to her bed, and remained unconscious for four days. During the first day of unconsciousness she was flaccid. This gave way to a typical picture of decerebrate rigidity which lasted three days. Consciousness then returned rather promptly and she has gone on to a complete recovery. Presumably the anoxia produced cerebral edema, not sufficiently severe to damage the cells permanently.

Results in those patients who survived surgery have been extremely gratifying. The children have been relieved of invalidism due to constant anoxia and the parents have been freed from the anguish of being unable to relieve the distress of their children. The first child on whom an aortic-pulmonary anastomosis was performed was 21 months old at the time of operation, Sept. 13, 1946. Before surgery she was totally helpless, had never tried to stand, was extremely cyanotic, and lapsed into unconsciousness repeatedly each day on the slightest exertion. She now plays and runs like other children, rides her tricycle and remains free from cyanosis. It must be remembered, however, that although these children can live fairly normal lives after successful surgery, they will always be handicapped by the irremediable pathology in their hearts.

REFERENCES

1. Blalock, A., and Taussig, H. B.: The Surgical Treatment of Malformations of the Heart in Which There Is Pulmonary Stenosis or Pulmonary Atresia, J. A. M. A. **128**: 189, 1945.
2. Potts, W. J., Smith, S., and Gibson, S.: Anastomosis of the Aorta to a Pulmonary Artery in Certain Types of Congenital Heart Disease, J. A. M. A. **132**: 627, 1946.

DISCUSSION

DR. ROLLINS HANLON, Baltimore, Md.—Dr. Blalock is extremely sorry he cannot be here to discuss this very interesting presentation by Dr. Potts.

Only one side-by-side anastomosis has been done in the Johns Hopkins Hospital. The patient was 10 months old. The operation went very smoothly, and the postoperative course has been very gratifying.

A total of 415 cyanotic patients suspected of having pulmonary stenosis have been operated on by Dr. Blalock and his associates. The total mortality was 18 per cent. This includes all deaths, whether in or out of the hospital, and also those cases in which the diagnosis was in error. These latter included transposition of the great vessels and the Eisenmenger complex.

When it has been possible to do an anastomosis between the subclavian artery and the side of the pulmonary artery, the mortality has been approximately 10 per cent. There have been 283 such cases. Most of the cases that have survived the operation have done remarkably well; complications have been quite infrequent.

There have been a few instances of cardiac failure. One case developed subacute bacterial endocarditis, but this yielded readily to penicillin therapy.

Recently we have employed angiocardiography as an additional aid in investigation of these cases. This has been done in thirty-five cyanotic patients. More than half of these patients showed the typical tetralogy of Fallot; some of the others comprised transposition of the great vessels, truncus arteriosus and Eisenmenger's complex.

In association with electrocardiography and other physiological studies, we have found angiocardiograms to be of considerable aid in the diagnosis of some of the more obscure cases.

We have employed seventy-five injections of Diodrast without any untoward results to date.

DR. WILLIS J. POTTS, Chicago, Ill.—The clamp is available from Bruno Richter at 843 Duane Avenue, Glen Ellyn, Ill., or from V. Mueller and Company, 408 South Honore Street, Chicago, Ill. If you should order one, you will receive the middle-sized clamp which is suitable for practically all cases. The clamp is made in three sizes—small, middle, and large. We have used the middle-sized clamp on all but two cases; the small clamp was used on a child 4 months of age and the large clamp was used on a child 12 years of age.

A STUDY OF VARIATIONS OF THE BRONCHOPULMONARY SEGMENTS OF THE RIGHT UPPER LOBE

J. G. Scannell, M.D.,* Boston, Mass., and E. A. Boyden, Ph.D., Minneapolis, Minn.

THE purpose of the present article is to correlate and supplement the information obtained from dissection of fifty preserved specimens of the right upper lobe[4] with that obtained by injecting fresh specimens and subsequently dissecting them. The work thus represents a continuation of the program initiated with the left upper lobe.[9]

By detailed dissection of the fifty preserved specimens we were able to establish the prevailing bronchial, arterial, and venous patterns of the lobe and thus to identify the major variations; but obviously this method alone failed to provide sufficient information regarding the surface distribution of the segments or to correct the distortion which is inevitable in collapse and preserved specimens.

Concurrently, therefore, thirteen fresh adult lobes were injected according to a method previously described.[9]† Without repeating this in detail it is sufficient to note that camera lucida records were made of the segmental surface areas on the injected specimens and of the dissected bronchial tree in situ. Of the thirteen specimens, three have been selected for publication and three views of each presented: (1) the dissected mediastinal aspect of the lobe (corresponding to the lateral bronchogram); (2) the costal surface; and (3) the bronchial tree from the anterior, "surgical" aspect.

In discussing the specimens, the nomenclature of Jackson and Huber[7] has again been used, supplemented by a system of enumeration described by Boyden[3, 4] to correlate bronchi, arteries, and veins.

THE PREVAILING PATTERN

The specimen depicted in Fig. 1 of this paper has been selected to demonstrate the "prevailing" pattern of the right upper lobe. In our series of fifty dissections we are in agreement with Brock,[5] Foster-Carter,[6] Jackson and Huber,[7] and Appleton,[1] in finding that the right upper lobe is best described as dividing into three segments: apical, anterior, and posterior. (Apical, pectoral, and subapical—Brock; apical, pectoral, and posterior—Appleton; apical, anterolateral, and posterolateral—Foster-Carter.) This should not imply, however, that in the majority of cases a clear-cut trifurcation of the right upper lobe stem into apical, anterior, and posterior trunks will be found. In fact, in a series of fifty specimens the specific trifurcate arrangement alluded to was found in only 38 per cent, although a trifurcation of some kind was found in a total of 46 per cent.[4] However, since the remaining 54 per cent, although

From the Department of Anatomy, University of Minnesota.
Received for publication, November 26, 1947.

*Fellow in the Medical Sciences, National Research Council.

†In the course of the present study we have found that a 5-10 per cent formalin solution. containing 1 c.c. of 2 per cent aniline dye per 100 c.c. more satisfactory than the 15 per cent gelatin solution previously employed.

bifurcate, exhibited some ten different combinations of segmental and subsegmental bronchi, a trifurcate specimen has been selected to demonstrate the prevailing pattern (Fig. 1).

Here it will be noted that the apical segmental bronchus, B^1, divides into an apical (B^1a) and an anterior ramus (B^1b). Either, or both, of these may give rise to a lateral branch. The anterior segmental bronchus, B^2, underlies the anterior crest of the lobe (cf. *C.a.*, Fig. 3) in much the way that the lingular bronchus underlies the lingular crest in the left upper lobe.* This anterior segmental bronchus divides into a posterior ramus, B^2a, which finds representation along the secondary fissure, and an anterior ramus, B^2b, which supplies the region of the anterior angle of the lobe. The posterior segmental bronchus, B^3, divides into an apical, B^3a, and a posterior, B^3b, ramus. Among the criteria which we have found useful in identifying B^3b, which frequently has been found to arise independently of B^3a, is its relation to the interfissural crest. This will be discussed below.

Brock has very clearly shown the usual surface areas of bronchial distribution in the right upper lobe, and has also demonstrated in his plates the great variation in extent of the bronchopulmonary segments. The latter demonstration has been a stimulus to the present study, for it has seemed worth while to record for a given specimen both the surface pattern and the bronchial tree that gave rise to it, to the end that variations might be more fully understood.

Fig. 1 demonstrates the usual disposition of the three bronchopulmonary segments on the costal surface, namely, almost equivalent apical, anterior, and posterior territories. In only one of the thirteen injected right upper lobes could it be demonstrated that B^3a (of the posterior segmental bronchus) clearly entered the apex, although dissection of a much larger number of specimens had indicated that it appeared to enter the apex in 26 per cent.†

In the injected specimens it was found that the anterior ramus of the apical segmental bronchus (B^1b) frequently ventilated part of the apex and extended along the anterior margin of the lobe for a varying distance. The "normal" limits of this extension will be discussed under a subsequent heading. In contrast to the left upper lobe, no one segment appeared to be "dominant."

VARIATIONS IN THE BRONCHIAL PATTERN

In the series of fifty right upper lobes studied by detailed dissection which this study is intended to supplement, three striking and relatively common variations of the bronchial pattern were found.[4] These were (1) the development

*As on the left, certain ridges of the mediastinal and inferior surfaces of the lobe are prominent and constant enough to warrant designation. Due to the presence of the middle lobe, there is an additional elevation on the right side. This we have called the interfissural crest (Fig. 3). The four ridges are named as follows: (1) *Crista subclavia* (C.s.), which lies between the subclavian groove anteriorly and the groove formed by the vertebral bodies posteriorly; (2) *Crista posterior* (C.p.) formed by the junction of the interlobar and the posterior costal surface; (3) *Crista interfissuralis* (C.i.), formed by the junction of the interlobar and secondary fissural surfaces; (4) *Crista anterior* (C.a.) formed by the junction of the secondary and anterior mediastinal surfaces.

†In this one specimen there was some question of segmental displacement about the apex as a result of minimal tuberculous scarring about the B^1b bronchus. The apex is here defined as that portion of the lung which lies above the plane of the first rib.

In a comparable series of thirteen injected left upper lobes, B^3a was found to enter the apex in four instances.

EXPLANATION OF PLATE

(Sketches rendered for publication by Lawrence B. Benson)

The plate depicts a series of injected right upper lobes designed to show typical variations of the bronchopulmonary segments. Each specimen is represented by a view of the medial surface of the lobe, its costal surface, and a mediastinal view of the bronchial tree (apex to the left). The specimens have been drawn with the source of light to the upper right. For identification of the ridges on the medial surface, see Fig. 3.

Bronchial pattern:

B^1, apical segmental bronchus
- B^1a, apical ramus
- B^1b, anterior ramus

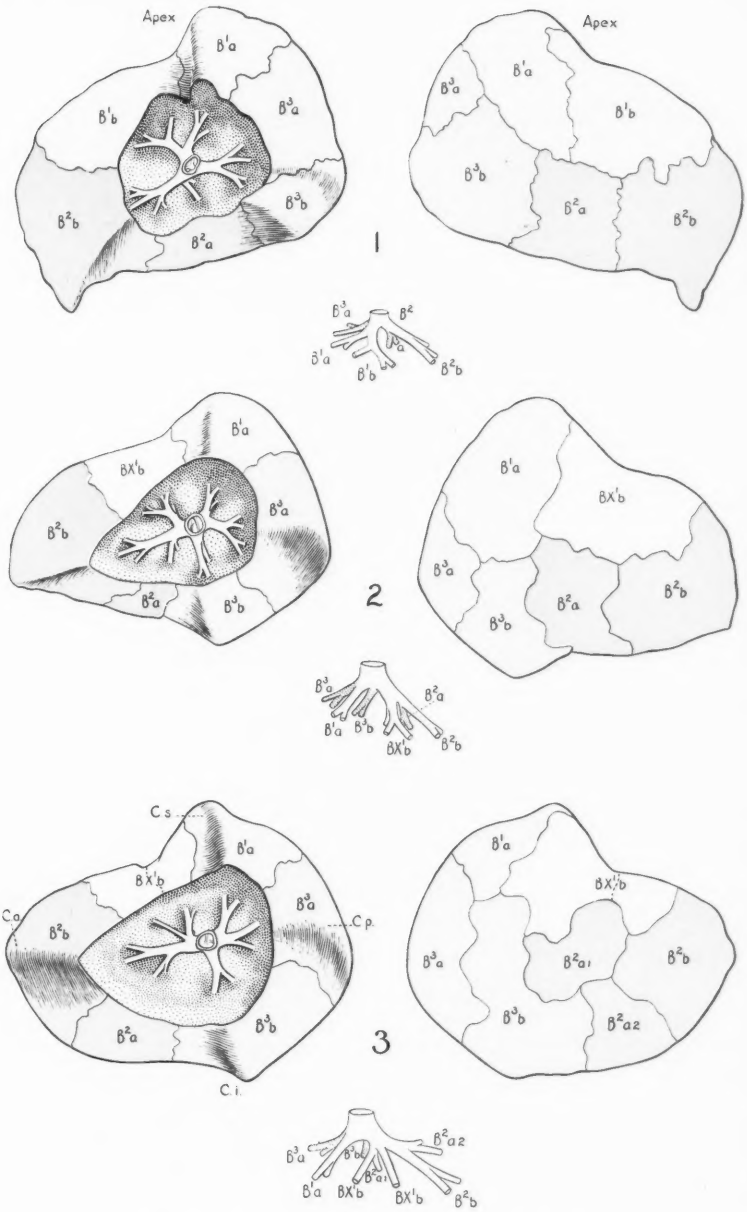
B^2, anterior segmental bronchus
- B^2a, posterior ramus
  - B^2a1, superior branch
  - B^2a2, inferior branch
- B^2b, anterior ramus

B^3, posterior segmental bronchus
- B^3a, apical ramus
- B^3b, posterior ramus

Fig. 1.—Injected specimen (No. 32) with trifurcate upper lobe bronchus, illustrating (1) the prevailing pattern of the lobe, and (2) some anterior displacement and spreading of the B^1 segment.

Fig. 2.—Injected specimen (No. 26) with V-shaped trifurcation, illustrating (1) an accessory anterior bronchus, BX^1b, (2) the separate origin of the components of the posterior segment, B^3a and B^3b. The latter arises coordinately with B^2 and therefore represents a transition to the "quadrivial" type of branching found in 16 per cent of dissected lobes.

Fig. 3.—Injected specimen (No. 21) with bifurcate bronchus illustrating a stage in the separation of the components of B^3. Here B^3b arises coordinately with bronchus $B^{1+3}a$. There is also an accessory anterior bronchus, BX^1b. *C.a.*, Crista anterior; *C.i.*, Crista interfissuralis; *C.p.*, Crista posterior; *C. s.*, Crista subclavia.



Figs. 1, 2, and 3.—(*For legend, see opposite page.*)

of an accessory apical-anterior bronchus, BX^1b (28 per cent); (2) an anterior rotation or spreading of the apical segmental bronchus (14 per cent); and (3) the separate origin of the two components of the posterior segmental bronchus, B^3a and B^3b (20 per cent).

1. *The Accessory Apical-Anterior Bronchus, BX^1b.*—The specimens depicted in Figs. 2 and 3 have been selected to demonstrate, among other things, the development of a major accessory bronchus, BX^1b. The term "X" has been applied to a bronchus which arises as an additional or accessory branch of one segmental bronchus to supply a territory usually served by the ramus of another segmental bronchus. Thus, in Fig. 2 it can be seen that the segmental area of the lobe usually supplied by the anterior branch of the apical segmental bronchus, B^1b, is supplied, in this instance, by a large branch of the anterior segmental bronchus, B^2. This branch has been designated BX^1b.*

Similarly, in Fig. 3 the anterior-apical zone, extending well into the apex, is supplied by BX^1b, the accessory branch of the anterior segmental bronchus. In this specimen BX^1b gives off a branch that ventilates an area usually supplied by the superior branch of the posterior ramus, B^2a1. Accordingly, the designation of B^2a1 has been preserved. The rest of the B^2a ramus (the inferior branch, B^2a2) arises "normally" from the anterior segmental bronchus. (In the left-hand sketch of Fig. 3, B^2a2 is labeled B^2a, the final "2" having been inadvertently omitted.)

2. *The Anterior Rotation or Spreading of the Apical Segmental Bronchus.*—Although dissection of fifty specimens has shown that in 14 per cent of a relatively large number of lobes, one might expect to find B^1b extending far enough down toward the anterior angle of the lobe to take over the territory usually supplied by the upper branch of B^2b (cf. Boyden and Scannell, Plate I, Figs. e. and f.), none was found in the present series of thirteen injected specimens. However, Fig. 1 of this article may serve to demonstrate the trend of the variation, in that the area served by the anterior branch of the apical bronchus extends well along the anterior margin of the lobe. However, it does not reach the point which, on the basis of a relatively large number of dissections, we have set as the "normal" inferior limit of B^1b—namely, a point two-thirds of the way from the apical-anterior notch to the inferior angle of the lobe.† When B^1b extends below this point, it takes over the territory supplied by B^2b1, and that ramus is absent. A surface view of such an anomalous spreading of the apical segment may be seen in Fig. 19*c*. of Brock's monograph.[5]

3. *The Separate Origin of the Components of the Posterior Segmental Bronchus, B^3a and B^3b.*—The third striking variation found in the analysis of

*It is interesting to compare Fig. 2 of this paper with Brock's figure 19*a* (p. 24).[5] In the latter a small apical segment is recorded as occupying an anterior-apical zone while most of the apex is occupied by an expanded posterior segment. The pattern of the two figures agrees so well in essentials that it seems likely that what he has called the apical bronchus—probably on the basis of its being the more anterior of two main ascending trunks—we would call an accessory branch of the anterior segmental bronchus. The true apical bronchus, B^1a, as determined by the area ventilated, would seem to arise in conjunction with part of the posterior segment.

†The notch in question is that indentation which is caused by the first rib and marks the transition between the apex and the anterior margin of the lobe. Although in many specimens it may be a relatively shallow incisure, it is generally definite enough to serve as a reference point.

fifty dissected specimens was the separate origin of the components of the posterior segmental bronchus. (This occurred in 20 per cent of the lobes dissected.) The specimen depicted in Fig. 2 demonstrates this variation. In this instance the upper lobe bronchus bifurcates, although a study of the keel in the orifice shows that the branching is a modification of the type designated by Appleton[1] as a "V-shaped" trifurcation, i.e., more like the type found in the left upper lobe. The three components have been interpreted to be $B^1a^{+3}a$, B^3b, and B^2 (with its accessory branch BX^1b). If the branching of the lobar bronchus be strictly considered as a bifurcation, then B^3a goes with the B^1a half, and B^3b with the B^2 component. The criterion which identifies this bronchus as B^3b and not B^2a is the position of the main axis of B^3b in the prevailing pattern; for in fifty-four of the sixty-three specimens (fifty dissected and thirteen injected lobes) in which this anomaly did not occur—i.e., separate origin of the B^3 components—the main axis of B^3b lay posterior to the interfissural crest.* Such is the position of the bronchus labeled B^3b in Fig. 2. On this basis, therefore, it could not be considered a B^2a. Furthermore, there is a recognizable B^2a already present in this specimen. Third, in this group of thirteen injected specimens and in the fifty dissected specimens, B^2a has never been found arising coordinately with B^2b at the level of the lobar bronchus.

Another possible interpretation would be to identify the B^3b in question with an independent lateral (or axillary) segmental bronchus which has been described by several earlier writers and more recently by Lucien and Beau.[8] However, in our own and other extensive recent series of dissections the occurrence of such a relatively independent "lateral" segment has been found to be uncommon.[1, 4, 5] Although the clinical implications of the term "lateral" or "axillary" segment might be useful, logically one would have to give separate names to any of a number of subsegmental bronchi whenever they arise at the level of the lobar bronchus. Furthermore, the "axillary" area, as Brock has emphasized, normally receives contributions from two segments, B^2 and B^3, in varying proportions.

Comparison of Figs. 2 and 3 reveals another interesting point in the arrangement of the bronchi of the right upper lobe. While B^3a and B^3b arise independently of each other in both these specimens, they have different relations to adjacent bronchi. In Fig. 3, B^3a is joined with B^1a and forms an apicoposterior trunk suggestive of the left upper lobe. In Fig. 2, however, B^3b has been displaced downward to a degree rarely, if ever, found on the left side; consequently, the specimen represents a form transitional to a new pattern which we have described in 16 per cent of right upper lobes. This is the "quadrivial" type of branching in which B^1 (or B^1a) arises coordinately with B^3a, and B^3b with B^2, the two pairs of orifices being separated by only a shallow keel (cf. Boyden and Scannell, Plate I, Figs. *f* and *i*). The separation of the two components of B^3 thus leads to one of the most striking variations in the bronchial pattern.

*To be sure, its surface branches may overlap the crest anteriorly. Thus in ten injected specimens, the crest lay within the territory of B^3b, and in two others within the area of B^2a. In the remaining one, associated with an extremely small middle lobe, no crest could be demonstrated.

SUMMARY

A series of thirteen right upper lobes has been studied by differential injection and subsequent dissection of the segmental bronchi. The prevailing pattern of the bronchopulmonary segments, namely, apical, anterior, and posterior, has been described and demonstrated. The three common variations of the segmental pattern have been discussed and illustrative specimens presented. These variations are: (1) the occurrence of an accessory apicoanterior bronchus, BX^1b; (2) the anterior rotation or spreading of the apical segment; (3) the separate origin of the component bronchi of the posterior segment, leading toward quadrivial branching of the right upper lobe bronchus.

REFERENCES

1. Appleton, A. B.: Segments and Blood Vessels of the Lungs, Lancet **2**: 592, 1944.
2. Appleton, A. B.: The Arteries and Veins of the Lungs, I. The Right Upper Lobe, J. Anat. **79**: 7, 1945.
3. Boyden, E. A.: The Intrahilar and Related Segmental Anatomy of the Lung, Surgery **18**: 706, 1945.
4. Boyden, E. A., and Scannell, J. G.: An Analysis of Variations in the Bronchovascular Pattern of the Right Upper Lobe of Fifty Lungs, Am. J. Anat. **82**: 27-74, Jan., 1948.
5. Brock, R. C.: The Anatomy of the Bronchial Tree, London, 1946, Oxford University Press. (Also in Guy's Hospital Reports, 1942-44, vols. **91, 92,** and **93.**)
6. Foster-Carter, A. F.: The Anatomy of the Bronchial Tree, Brit. J. Tubere. **36**: 19, 1942.
7. Jackson, C. L., and Huber, J. F.: Correlated Applied Anatomy of the Bronchial Tree and Lungs With a System of Nomenclature, Dis. Chest **9**: 1, 1943.
8. Mattei, C., Tristani, M., and Barbe, A.: Aspect radiologique pénicillo thérapie endobronchique des abcès pulmonaires, Presse méd. **54**: 638-639, 1946. (Quoting Lucien and Beau.)
9. Scannell, J. G.: A Study of Variations of the Bronchopulmonary Segments in the Left Upper Lobe, J. THORACIC SURG. **16**: 530-537, 1947.

COLLATERAL VENTILATION

P. R. Baarsma, M.D., and M. N. J. Dirken, M.D.
Groningen, Holland

INTRODUCTION

In an early period of histology, doubt arose as to the uninterrupted lining of the alveoli (Addison,[1] Rayney,[2] Bakody,[3] Donders,[4] Eberth,[5] and Virchow[6]). Henle[7] drew attention to the fact, that, as a result of this incomplete lining, communications exist between adjoining alveoli by means of "kreisrunde, scharfrandige Oeffnungen" (circular, sharp-edged apertures). On the other hand, with the help of extensive photographical material, Koelliker[8] put forward the opinion that the interstices between the capillaries of the lungs were filled by irregularly formed flat cells, extending over the capillaries themselves. According to Bargmann,[9] this work of Koelliker has been the foundation for all that was taught in hand- and textbooks concerning the lining of the alveoli, for decades.

In the meantime, Kohn[10] showed (with pneumonia patients) the occurrence of fibrinous threads running right through the alveolar wall. In 1894, Miller,[11] from his extensive anatomic studies, concluded that alveolar stomata, if found, are artefacts arising from fixation or by some other means. To this view Laguesse[12] and Oppel[13] later subscribed. At this same time, however, Hauser,[14] Hansemann,[15] Nicolas,[16] Merkel,[17] and Schultze[18] found distinct apertures in the alveolar wall of human beings and various mammals. In 1929, Macklin[19] published an investigation in which he denied the existence of these apertures, but in 1934, on the strength of extensive investigations making use of the latest technical developments, he came over to the opposite view. This author investigated the lungs of human beings from 2½ to 80 years of age, and found the "alveolar vents" much more frequent than had been assumed. Adams and Livingstone,[20] Joannides,[21] and Seemann[22] later confirmed the occurrence of alveolar stomata in mammals and in man, the latter author suggesting the possibility that these stomata might possess some physiologic significance.

The morphologic observations, which indicated the existence of communications between adjoining alveoli, found confirmation in clinical observations and in the results of experiments by van Allen and his collaborators.[23-28] On the clinical side, van Allen, Coryllos,[29-32] and Huizinga[33] have pointed out that, upon obstruction of a bronchus, atelectasis occurs only when access to an entire pulmonary lobe is obstructed in this way. If the obstruction affects only part of a lobe, no atelectasis occurs; therefore, the air must have some roundabout way left by which to enter the obstructed part of the lung. Experiments by van Allen, Lindskog, and Richter and by Kourilsky and Anglade[34] on experimental atelectasis have brought confirmation of this, but have, at the same time,

From the Oto-Rhino-Laryngological clinic, Professor Dr. Eelco Huizinga, director, and the Physiological Laboratory, Dr. M. N. J. Dirken, of the University of Groningen, Holland, temporary director.
Received for publication March 28, 1947.

made it clear that atelectasis fails to appear only when no inflammation occurs in the obstructed part. Any inflammation, however small, is sufficient to stop the collateral access of air and make possible the development of an atelectasis.

Of the experiments described by van Allen, the most impressive is that which he carried out in collaboration with Jung.[24] A catheter was introduced in a dog, obstructing the bronchus of the lower lobe just past the first bifurcation. The free end of the catheter was kept under water. At every inspiration, water was sucked about 5 cm. into the catheter. At every expiration, however, air bubbled out of the opening of the catheter, and this occurred as long as the experiment lasted. If, on the other hand, the catheter was introduced in such a way as to make the obstruction localized before the first bifurcation of the bronchus of the lower lobe, so that the entire lobe could only ventilate through the catheter, air escaped from the immersed end only once. This, indicated, therefore, that a collateral access of air, outside the obstructed bronchus is possible. A necessary condition is, however, that there is a free communication of part of the lobe with the air outside.

As the existence of the alveolar stomata has been criticized from histologic quarters, so there exists also a possibility that the collateral access of air, here described, is only an artefact. The wall of the alveoli, in between the capillaries, is, doubtless, extremely thin, and proof only against very slight pressure. In his animal experiments, van Allen found that under normal inspiration in the obstructed part of the lobe, a pressure of –5 cm. water occurred. In the clinical cases in which obstruction by a foreign body must have caused a strong tussal stimulus, the difference in pressure between the obstructed and the non-obstructed lung tissue must have been temporarily still much greater. It can be imagined readily that the delicate alveolar wall may break down in some places. All this, however, does not diminish the great importance that collateral access of air undoubtedly can occur in pathologic cases.

Under normal respiratory conditions, the difference in pressure between the outside air and the air in the lungs, according to the calculations of Rohrer[35] and according to the experiments of Bayliss and Robertson[36] amounts to only a few millimeters of water, so the difference in pressure between contiguous lobuli must be still less.

In order to ascertain whether, during normal respiration, the existence of a collateral ventilation by the side of bronchial ventilation of the lungs must be assumed, it is necessary to keep the differences in pressure on both sides of the alveolar wall extremely small. Starting from van Allen and Jung's experiments described above, we have tried to verify in this way the existence of collateral ventilation during normal respiration.

METHOD

The experiments were made on rabbits in urethane narcosis (1 gr. per kg.). The small diameter of the bronchial tubes of these animals precluded the use of an inflated catheter, as used by Coryllos and others. At the end of a semiflexible catheter *c* (Fig. 1) a hollow cylinder of laminaria *a* was fixed by means of a copper ring *b*. On introduction into the bronchus, the laminaria-cylinder absorbed moisture and began to expand, and the part under *b*, being unable to expand, acquired a funnel shape. The free edge of the funnel

pressed into the mucous membrane and provided an airtight fitting; at the same time, however, the opening of the catheter was rendered free of the wall, so that there could be no chance of its getting choked up. The catheter was introduced by means of a tracheotomy opening, with the help of the bronchoscope, after Brünings. When the end of the catheter had been brought to the place determined beforehand, a few minutes' interval was allowed for the laminaria cylinder to swell, in order to fix the catheter, then the bronchoscope tube was withdrawn. On completion of the experiment, both the position and the fitting were variably examined in the excised lungs.

By means of the catheter *A* (Fig. 2) and the rubber-tube fixed to it, the part of the lung thus cut off was connected with the air chamber of the bottle *B*, which was partially filled with water. Further connections existed between this chamber and the optic manometer *F*, and the electromagnetic valve *D*, the latter regulating the connection with the volume-recorder F. The manometer, recently described by us,[37] had an excursion of 1 cm. for 0.38 cm. water pressure, the volume recorder gave 2.1 cm. on the photographic paper for every cubic centimeter.

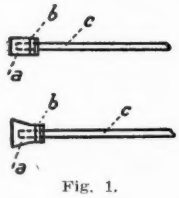


Fig. 1.

If, on inspiration, the valve *D* were shut, a negative pressure would develop in the system, the height of which would, breath excursions of the test object being equal, depend on the volume of air present in the bottle *B*. This volume was adjusted before each experiment by means of the bottle *C*. Now, if, at the beginning of expiration, valve *D* were opened, the volume-recorder would be sucked in if pressure in the system were still below the atmospheric pressure, but after that it would rise to the original position. If, however, collateral ventilation did exist, air must have entered during inspiration, as a result of the difference in pressive between the cut-off part of the lung and its surroundings, and during expiration when the pressure in the system equaled that of the atmosphere, this air must have escaped toward the volume recorder, *S*. For a quantitative measurement of the extent of collateral ventilation, it was necessary that the valve *D* be shut exactly at the beginning of each inspiration and opened at the beginning of each expiration. For this purpose, the abrupt fluctuations which the negative thorax pressure shows at the beginning of in- and expiration lent themselves excellently.

For this, the pleura cannula, as described by Dirken and Heemstra,[38] was used mostly in the right half of the thorax and connected with the water manometer *G*. In the short leg of this manometer lay a small plug of cotton wool *h*. This plug, pressed upward or downward by the very first movements of the water column on inspiration and expiration, closed or opened a mercury contact by means of a lever. The contact point had an excursion of only a few millimeters; the plug of cotton wool was small enough to let the water pass at a further excursion of the water column. Through the interconnection of the relay *M*, the valve *D* was operated by the mercury contact.

RESULTS

A. *Model Experiments.*—In order to get an insight into the value of various factors that may play a part in collateral ventilation, a number of model experiments were made. For this purpose the same apparatus was used, but the experimental animal was replaced by a small, electrically driven pump, while

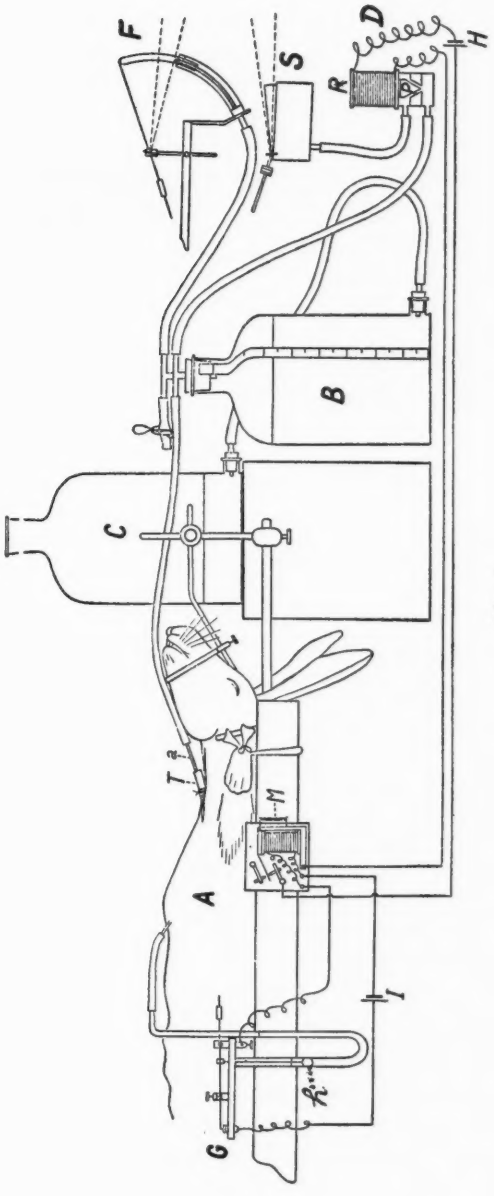


Fig. 2.

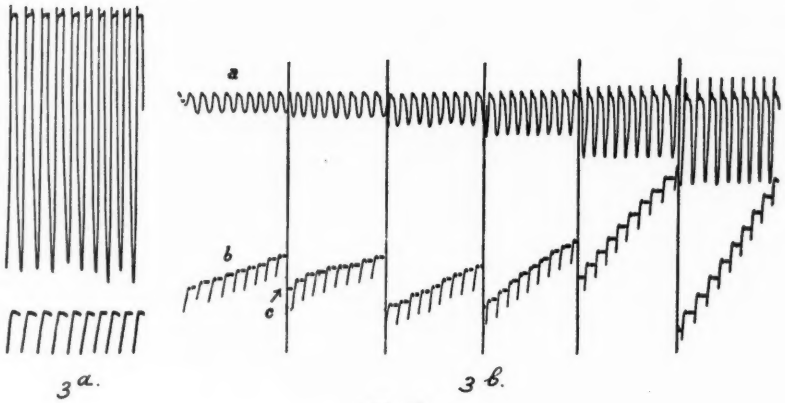


Fig. 3.

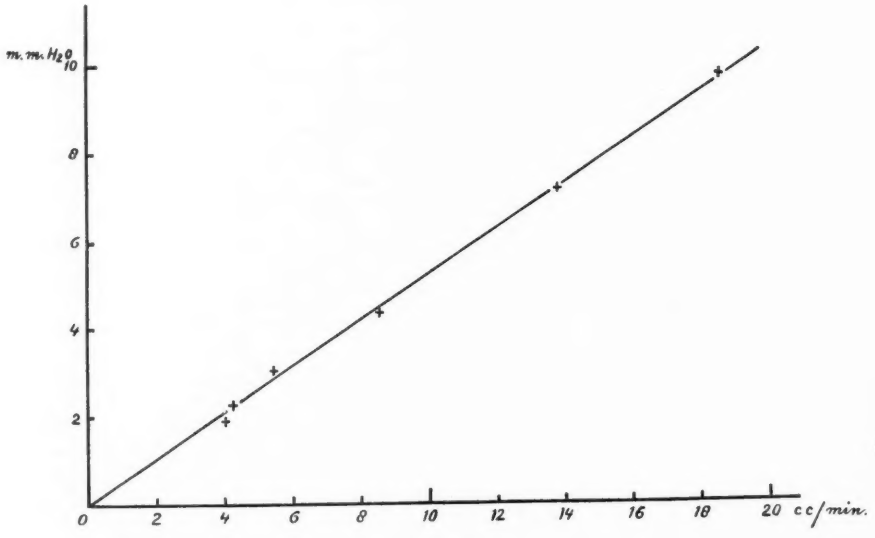


Fig. 4.

the valve *D* was operated by means of a sliding contact on the crankshaft of the pump. Fig. 3, *A* gives a representation of a curve thus recorded, which was taken with a small air content of the bottle *B*, so that large fluctuations of pressure were the result (upper curve). As the quantity of air in the system does not increase or decrease, the volume recorder (lower curve) remains at the same level. This condition would exist, according to van Allen, in the ventilation of the pulmonary lobes, as these do not possess a communication with other parts of the lung outside the bronchi.

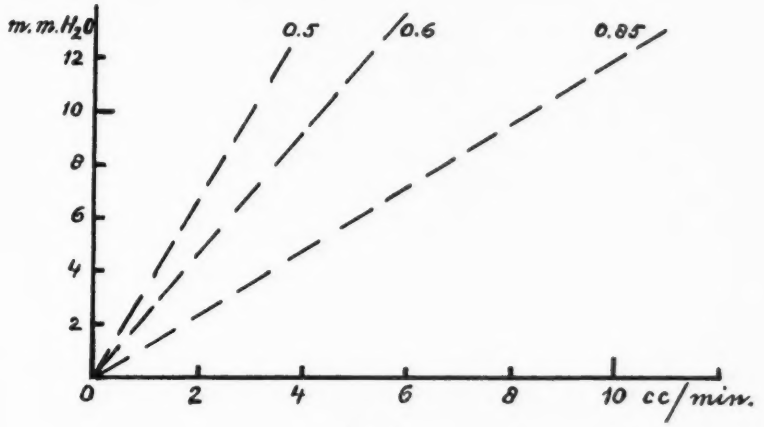


Fig. 5.

The collateral ventilation was imitated by placing a capillary side tube between the pump and the end of the catheter, so that by this narrow communication the system was in contact with the air outside. A number of recordings were made with differing air contents of the bottle *B*. In Fig. 3, *B*, the decreasing air content of *B* from the increase of the negative inspiration pressure in successive experiments may be seen. At the same time, it is evident, however, that more air is sucked in collaterally through the capillary as the negative pressure during inspiration increases. When the values obtained from this curve are set down in a diagram (Fig. 4), there appears to be a rectilinear correlation between the height of the negative pressure prevailing in the system and the quantity of collaterally sucked-in gas. Increasing the frequency from thirty to sixty strokes a minute gives no changes in the quantity of air sucked in collaterally. An increase of the stroke volume causes an increase of the quantity of gas sucked in collaterally, because with one and the same content of the bottle B the height of the inspiratory negative pressure depends on the stroke volume. The collateral gas supply is mainly determined by the resistance (by the diameter of the capillary used) as appears from Fig. 5, where capillaries of a diameter of 0.5, 0.6, and 0.85 mm. have been compared.

From this figure it is clear, moreover, that if the resistance of the communication should change with the differences in pressure, the resultant line should not be straight but curved.

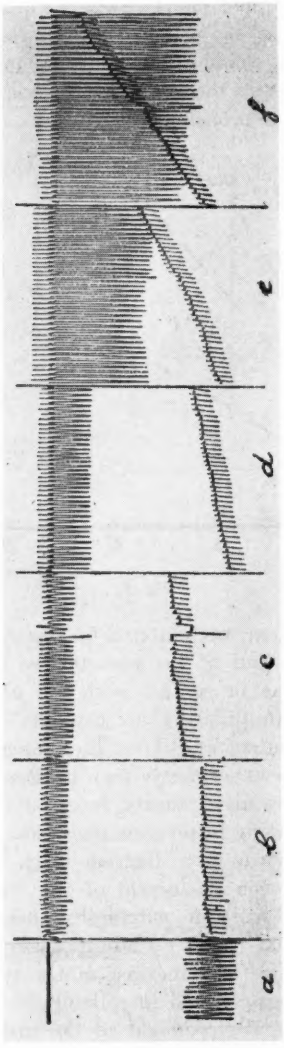


Fig. 6.

B. *Animal Experiments.*—In some twenty experiments, the laminaria end of the catheter was introduced into either the right or the left bronchus of the lower lobe, past the upper bifurcations to the lower lobe with the help of a bronchoscope. After the laminaria funnel had expanded, the cut-off part of the lung was first connected directly with the volume recorder so as to ascertain the degree of ventilation (Fig. 6, *a*). Next, the catheter was connected with the apparatus represented in Fig. 2, and recordings were made, the air content of the bottle *B* being reduced each time (Fig. 6, *b-f*). It is evident that the content of the volume recorder increases more quickly as the difference in pressure developing during inspiration between the cut-off part of the lung and its surroundings increases. Since the apparatus was tested for leaks before

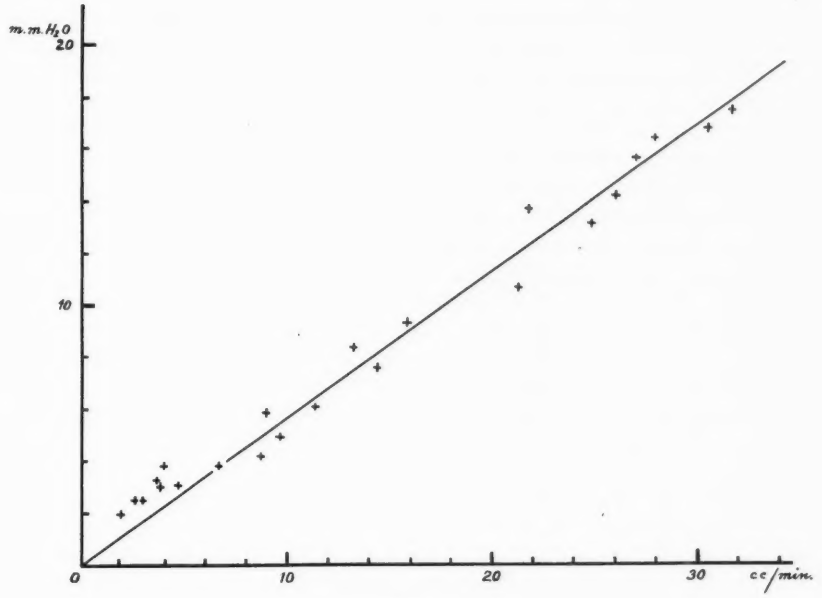


Fig. 7.

the experiment, and as the airtight fitting of the catheter in the bronchi was checked on its completion, the only possibility is that the communication is to be found within the lungs themselves past the end of the catheter. In Fig. 7 the quantity of air supplied collaterally per minute in such an experiment, has been entered against the corresponding inspiratory pressure. In all experiments, these points were found to be grouped about a straight line going through the zero point in most cases; in only a few cases a negative pressure of 2 mm. water was required before collateral suction occurred. Then, too, however, there existed a fixed proportion between gas supply and pressure.

In the individual experiments, a large difference in the incline of the line did exist, that is, a difference in the intensity of the collateral air supply at one

and the same pressure. This was to be expected, as it is probable that the extensiveness of the cut-off part of the lung and, therefore, the contact with the surrounding tissue, varied in the several experiments. In order to be able to compare the results in some measure, it was supposed that there is a relation between the size of the lung area connected with the catheter and its changes in volume during respiration. For this reason, the breath volume was registered both before and after each experiment (Fig. 6, *a*). It was found that the collateral air supply at a difference in pressure of 20 mm. water amounted to: 37, 39, 41, 33, 43, 36, 36, 44, 45, 37, 43, 40, 36, 44, 47, 42, 33, 34, 37, and 44 per cent, respectively, in the several experiments, with an average of nearly 40 per cent of the breath volume. When the slightly greater pressure difference of 35 mm. water was attained, the volume of the collateral ventilation even amounted to 65 to 66 per cent of the normal ventilation through the bronchus.

When the valve *D* was made to shut off the access to the volume recorder during expiration instead of during inspiration, by adjusting the relay *M* (Fig. 2), a positive pressure developed in the system during expiration. At every inspiration the volume recorder dropped, so that a discharge of gas by the collateral communications was also possible. This discharge turned out to be the exact image of the described increase when inspiration was obstructed.

An entirely different result was obtained if an entire lobe was connected with the system. In most cases the cardiac lobe was used for these experiments, a lobe which, in rabbits, lay entirely isolated in the mediastinum, against the bottom of the heart. For comparison, the entire lower lobe, either right or left, was obstructed in some experiments. Sixteen experiments were made in this way, all, except in cases to be discussed separately, yielding the same result. In none of these cases could any collateral access or discharge of gas be discovered, and both at lower and higher pressure the curve conformed to the model curve represented in Fig. 3, *a*. This negative result was of special significance, moreover, as in this way it was demonstrated that the fixing of the catheter in the bronchus and the rest of the apparatus was airtight, and that the rising or dropping of the volume recorder found on shutting off a part of the lobe did not arise from leakage but from communication in the lung tissue itself.

In three cases, however, notwithstanding that the entire cardiac lobe had been connected, an access of air, however slight, was observed. At a pressure of 20 mm. water, this amounted to only 2.7, 4.1, and 3.6 per cent of the breath volume, respectively. In view of the results already mentioned, this indicated the presence of connections with other lung tissue, and on histologic examination,* it turned out that in these three cases there existed a bridge of lung tissue between the cardiac lobe and the right lower lobe. In none of the experiments yielding a negative result was such a bridge of tissue discovered.

These results indicate that the component parts of a pulmonary lobe communicate outside the bronchi, and that a similar communication does not exist between the different pulmonary lobes themselves, unless these are connected by a bridge of lung tissue. Some experiments were conducted in order to ascertain whether the width of the collateral communications could be influenced.

*We owe thanks to Prof. de Haan for his ready help in this and the remaining histologic work in this investigation.

Cutting or stimulating the vagus and the sympathetic nerve had no effect, no more had the cutting of the phrenic nerve when pectoral respiration took over the function of the diaphragmal respiration normal for rabbits. In all these cases, the extent of the collateral air supply was determined solely by the negative pressure developing in the system during inspiration. This also held good for the respiration changes caused by breathing mixtures rich in carbon dioxide and those caused by a partial stenosis of the bronchial tubes.

DISCUSSION

The experiments here recorded confirmed the results obtained by van Allen and his collaborators concerning the existence of communications between the component parts of a pulmonary lobe. In the model experiments, it has been shown that the quantity of gas sucked in collaterally was directly proportionate to the negative pressure if the width of the capillary leak remained unaltered (Fig. 5). In the animal experiments, we found in the same way that the quantity of gas sucked in collaterally was directly proportionate to the negative pressure (Fig. 7) and from this we may conclude that here, too, the total width of the communications has remained unchanged. *From this, it follows that the communications meant cannot be fissures of tissue, because their permeability does not increase with increasing pressure.* The observation that these communications were open even at the most minute differences in pressure, leads to the conclusion that these permanent communications also have some significance for normal ventilation of the lungs. The ventilation of a lobulus of a pulmonary lobe may also take place apart from ventilation by its proper bronchus, by means of collateral ventilation from the adjoining lobuli.

This collateral ventilation will certainly come into operation if even minute differences in pressure arise in contiguous lobuli during respiration; in this way, a balance of pressure is effected, and at the same time the uniform constitution of the contents of a pulmonary lobe is promoted. From our results, it also appears that as regards its extent, this collateral ventilation may become very considerable. As has been stated, the collateral access of air at a difference in pressure of 2 cm. water, amounts to 40 per cent of the quantity that is normally supplied by the bronchi. This is true, while of the entire lower lobe, only the upper branch of the bronchus is connected with the outer air, so that the area of contact is small in proportion to the volume. This is the reason why rabbits, at least, may be expected to possess numerous open communications between the lobuli of a pulmonary lobe.

The existence of anastomoses between the finer ramifications of the bronchi has not been described in the literature, but, on the other hand, the histologists have occupied themselves with the existence of communications between adjoining alveoli. The existence of alveolar stomata has been confirmed recently by various investigators, although doubt still prevails as to the number and nature of these communications. Moreover, alveolar stomata have not yet been demonstrated in rabbits. We have examined the lungs of some of our experimental animals for them. Contrary to the usual way of fixation, and in order to preserve the normal condition as well as possible, we allowed the fixation fluid

(Bouin's solution) to flow into the lungs of the living animal at a pressure of not more than 2 cm. water. It was introduced by means of the catheter which, as in the above-mentioned experiments, shut off part of the lower lobe. If the fixation fluid was allowed to flow in at such slight pressure, the lung filled like

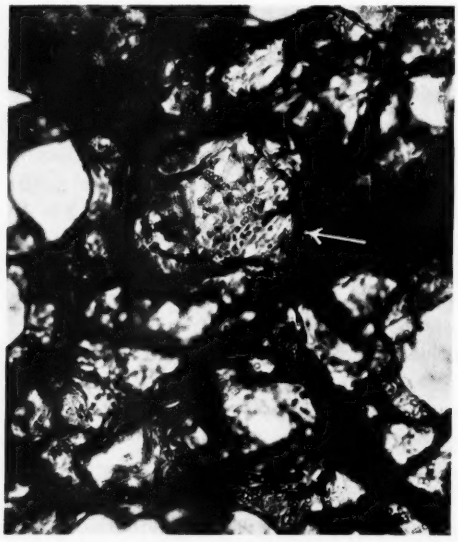


Fig. 8.

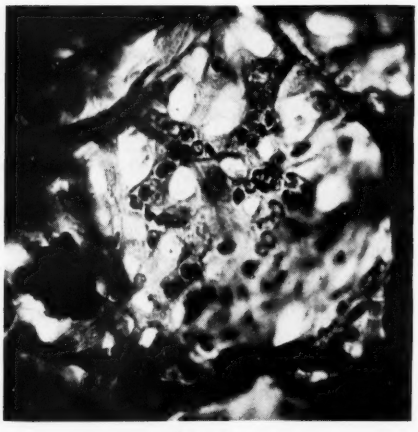


Fig. 9.

a sponge; the animal, who had been brought into a heavy narcosis, showed no reaction whatever, probably because both the circulation and the conduction of stimuli were interrupted as soon as the tissue came into contact with the fixation fluid. This also explains why the blood in the left heart was found

to be liquid and not at all discolored at autopsy. This way of fixation had the advantage that the normal expansion of the lungs was preserved and also the normal state of circulation; the pressure applied was so minute that the capillaries, too, remained filled with blood. After preliminary treatment and imbedding in celloidin, sections were cut 50 to 100 μ thick, much thicker than the usual sections. Coloring of the sections was effected by means of hematoxylin-eosin, orcein Mallory, or orcein van Gieson.

Fig. 8 shows a typical general picture of the lung tissue in the slides thus prepared. In the wall of all alveoli that lend themselves to a close observation are found sharply outlined openings which, however, vary greatly in both number and form. The alveolar wall, as may be seen in the alveolus that has been further indicated in the figure, often seems to consist merely of a mass of capillaries which may be clearly recognized as such from the blood cells present in them. This same alveolus has been reproduced much enlarged in Fig. 9. In Fig. 10, a single stoma of another alveolus has been brought into focus. It

Fig. 10.

appears clearly from this photograph that only capillaries form the border of the stomata. The fact that the stomata are only meshes in the capillary network is most clear in binocular observation, where the entire dome of the alveolar wall may be followed. The occurrence of numerous stomata in the lungs of birds and some minor mammals (bat) has already been described by others. Our sections show that an all-around ventilation of the lung capillaries with air exists also with larger mammals. That this could be demonstrated is probably due to the method of fixation in vivo, preserving the normal expansion and the normal filling with blood. Not much could be perceived of a spanning of the stomata by elastic fibers, as described by a few investigators (Macklin[19]). Upon staining with orcein, van Gieson, elastic fibers could be seen here and there, but their course was irregular and in no way connected with the limits of the stomata.

These stomata were found in every section. Their size varied and depended on the width of the meshes of the capillary network. Their form, too, varied much, being sometimes a true circle, sometimes not more than a slit. It is quite possible that their form is mainly determined by the filling of the lung capillaries with blood. Here and there a few cells could be found in the meshes of the capillary network which lay outside the capillaries.

From these microscopic observations it is clear that in the alveolar walls of rabbit lungs, a large number of stomata exist. These findings agree with the results of the experimental part of this investigation, and explain how a collateral ventilation of the lung tissue is possible. Outside the bronchi, the air can escape through the stomata from alveolus to alveolus and even further, as the partitions of connective tissue in between the acini, lobuli, and the larger parts of the lobes are no more than wide-meshed nets. The ample degree to which these interalveolar communications exist explains the large gas transport that could be demonstrated in our experiments and, at the same time, stresses the importance collateral ventilation must have for maintaining a balance of pressure in the lungs and for the uniform composition of the air in the various parts of a pulmonary lobe.

SUMMARY

In rabbits, all parts of a pulmonary lobe communicate outside the bronchi, so that transport of considerable quantities of gas is possible.

The width of the communications rendering this collateral gas transport possible is independent of the difference in pressure between two parts of the lobe, so that we must assume that the communications are open also under normal respiration conditions.

Through these open communications, collateral ventilation may be brought about during respiration, guaranteeing a balance of pressure in the various parts of a pulmonary lobe and promoting a uniform constitution of its content. Between the pulmonary lobes as such these communications do not exist, unless they are connected by a bridge of lung tissue.

From a review of histologic literature and from microscopic observations, it is rendered probable that the alveolar stomata must be considered as the communications responsible for the collateral ventilation.

REFERENCES

1. Addison, T.: Guy's Hosp. Rep. 1843.
2. Rayney, G.: Brit. and foreign med. chir. Rev. **16**: 491, 1855.
3. Bakody, Th.: Virchow's Arch. **33**: 264, 1865.
4. Donders: Physiol. des Mensch. Leipzig, 1859.
5. Eberth: Virchow's Arch. 24, 1862.
6. Virchow, R.: Virchow's Arch. **82**: 238, 1880.
7. Henle, J.: Handbuch Anat. des Menschen, Aufl. 1873.
8. Koelliker, A.: Sitzungsbericht Physik. Med. Ges. Wurzburg, 1880.
9. Bargmann, W.: Zeitschr. f. Zellforsch. **23**: 335, 1935.
10. Kohn: Munch. med. Wchnschr. **42**: 3, 1893.
11. Miller, W.: J. Morph. and Physiol. **8**: 165, 1893.
12. Laguesse, G. E.: Arch. per le sc. med. **51**: 45, 1927.
13. Oppel, A.: Lehrbuch Verg. Anat. Bd. 6, Jena, 1905.
14. Hauser: Munch. med. Wchnschr. I, 1893.
15. Hansemann, D.: Sitzungsbericht preusz. Akd. Wiss. Kl. **9**: 1895.
16. Nicolas, A.: Traité d'Anat. Humaine **4**: 393, 1898.
17. Merkel: Bardeleben's Handbuch d. Anat. 1902.
18. Schultze, Fr. E.: Sitzungsbericht preusz. Akad. Wiss. Kl. **6**: 225, 1906.
19. Macklin, C. C.: J. Anat. **69**: 188, 1934; Arch. Path. **21**: 202, 1936; Physiol. Rev. **9**: 1, 1929.
20. Adams, W. E., and Livingstone, H. M.: Arch. Surg. **23**: 500, 1931.
21. Joannides, M.: Arch. Int. Med. **47**: 19, 1931.
22. Seemann, G.: Beitr. z. Path. Anat. u. z. allg. Path. **81**: 508, 1929.

23. Allen, C. M. van, and Adams, W. E.: Surg., Gynec. & Obst. **50**: 385, 1930.
24. Allen, C. M. van, and Jung, T.: J. THORACIC SURG. **1**: 3, 1931.
25. Allen, C. M. van, and Lafield, W. A.: Radiol. **22**: 27, 1934.
26. Allen, C. M. van, and Lindskog, G.: Arch. Surg. **21**: 1195, 1930.
27. Allen, van, Lindskog, G., and Richter, H.: J. Clin. Investigation **10**: 559, 1931.
28. Allen, van, Lindskog, G., and Richter, H.: Yale J. Biol. Med. **2**: 297, 1930.
29. Coryllos, P. N.: Surg., Gynec. & Obst. **50**: 795, 1930.
30. Coryllos, P. N., and Birnbaum, G. L.: Arch. Surg. **14**: 1346, 1929.
31. Coryllos, P. N., and Birnbaum, G. L.: Arch. Surg. **18**: 15, 1929.
32. Coryllos, P. N., and Birnbaum, G. L.: Arch. Surg. **16**: 501, 1928.
33. Huizinga, E.: Ztschr. f. Hals- Nasen- Ohrenheilk. **48**: 17, 1941.
34. Kourilsky, R., and Anglade, P. H.: Bull. et mem. soc. méd. d. hôp. de Paris **52**: 249, 1936.
35. Rohrer: Handbuch d. Norm. u. Path. Physiol. **2**: 110,
36. Bayliss and Robertson: Quart. J. Exper. Physiol. **29**: 27, 1939.
37. Baarsma, P. R., and Dirken, M. N. J.: Acta Brevia Neerl. **13**: 1943.
38. Dirken, M. N. J., and Heemstra, H.: Acta Brevia Neerl. **12**: 14, 1942.

COLLATERAL VENTILATION IN MAN

P. R. Baarsma, M.D., M. N. J. Dirken, M.D., and Eelco Huizinga, M.D.
Groningen, Holland

INTRODUCTION

IN AN earlier communication by Baarsma and Dirken,[1*] it was demonstrated that there exists a collateral ventilation of the lungs in rabbits. This had first been described in dogs by van Allen and his collaborators.[2] When a side branch of a lobe of the lung is disconnected, this part is still ventilated to a considerable extent because the air can circulate via a normally operating bronchus and the alveolar vents. These vents, the existence of which has been established by many investigators, can be made convincingly visible by a special method of fixation. A quantitative determination of collateral ventilation was first made in rabbits. Collateral ventilation proved to appear even with very small differences in pressure between the connected and disconnected parts of the lung, that is, with pressure differences of slightly more than 2 mm. water. Collateral ventilation, therefore, must be of importance in physiologic as well as pathologic circumstances. The air content of a special part of the lung was measured with normal and with collateral ventilation. When a branch of the lung was obstructed, it appeared that there was an average ventilation of 66 per cent of the normal (a considerable percentage) due to collateral ventilation.

When collateral ventilation had been demonstrated in two mammals, the next step was to see if it existed in man. We had been suspecting this for a long time, because of certain patients who had been observed in the clinic. Patients with a foreign body in one of the deeper respiratory passages are of particular importance in this respect. These patients often become experiments in vivo, so to say, in which it may be assumed that suddenly a certain bronchus of a hitherto healthy lung is disconnected. The exact localization of the obstruction can be determined by means of radiologic examination and bronchoscopy. In the case of a complete obstruction of a main bronchus or a lobar bronchus, there is always atelectasis. We had, however, been struck by the fact that in some patients where the bronchus was obstructed at a deeper point, notably beyond the first division of the bronchus of the lower lobe, there was no atelectasis. There is thus a very sharp contrast between cases where atelectasis can, and cannot be demonstrated.

This is most obvious in the following cases, which have been the starting point of our investigations. In both patients studied a metal foreign body had been aspirated on the left side. These metal objects are especially suitable in these cases because they can be localized easily by radiography, and because they are not so likely to cause an inflammation and thus obscure the syndrome.

Received for publication April 24, 1947.

From the Laryngorhinological Clinic and the Physiologic Laboratory of the University of Groningen, Holland.

*Page 238, this issue.

CASE 1.—A girl 10 years of age aspirated a small metal object ten days previous to our examination. She had a violent fit of coughing, and kept coughing, with occasional dyspnea. The left thorax obviously lagged behind in respiration. In percussion, a strong dullness was noticed on the left side, especially over the lower lobe. On the left side the breath sounds had almost disappeared. On the radiograph of Fig. 1, the metal object was conspicuous on the left side; trachea and bifurcation were visible; the foreign body appeared to be located in the left lower section of the main bronchus and in the first part of the bronchus of the lower lobe. There was a strong dislocation of the mediastinum to the left, and to the right a "naked" vertebral column was visible.

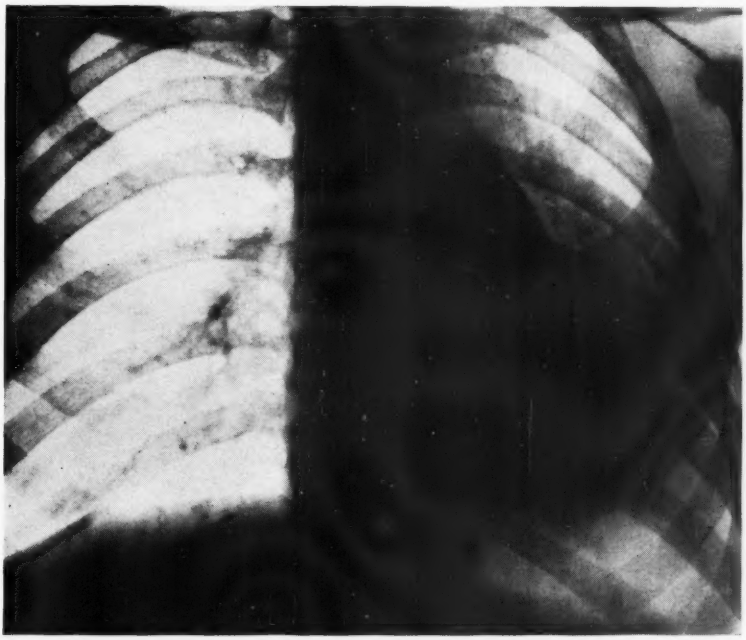


Fig. 1.

The radiologic localization of the foreign body was confirmed by bronchoscopy. It was located in the lower part of the main bronchus and the upper part of the bronchus of the lower lobe, and obstructed the whole of the lower lobe by its somewhat broader lower end. The object was extracted. The child recovered completely, after there had been symptoms of inflammation in the atelectatic left lower lobe.

The radiograph of Fig. 1 is very typical of an atelectasis of the left lower lobe, there is a very homogeneous shadow, the so-called "ground-glass shadow" of van Allen.

CASE 2.—A boy, 2 years old, had aspirated a metal pellet five days before examination. There was severe dyspnea immediately after the accident, which soon passed off. As the child kept coughing, he was sent to the clinic.

When admitted, the child did not appear to be ill. There was no dyspnea, *and the body temperature was entirely normal.* No deviations could be found by percussion, but the breath sound was weaker on the lower left side.

The pellet is clearly visible on the roentgenogram of Figs. 2 and 3. These radiographs have been taken during deep inspiration and deep expiration, respectively. The dislocation of the diaphragm is striking. *There is no trace of atelectasis and there are no symptoms of*

bronchostenosis. In both radiographs the mediastinum is in its normal position. During respiration it moves neither to the left, as may be expected in a case of total obstruction (Holzknecht's symptom), nor to the right, as is seen in valve stenosis.

It could be assumed on the strength of these roentgenograms that the foreign body was localized deep in the bronchial tree, in the bronchus of the lower lobe. This was confirmed by bronchoscopy. The pellet was located beyond the first dorsal branch of the bronchus of the lower lobe. There was no reaction of the mucous membrane and no sign of inflammation. Further on, the pellet hermetically closed the bronchus of the lower lobe; it was even

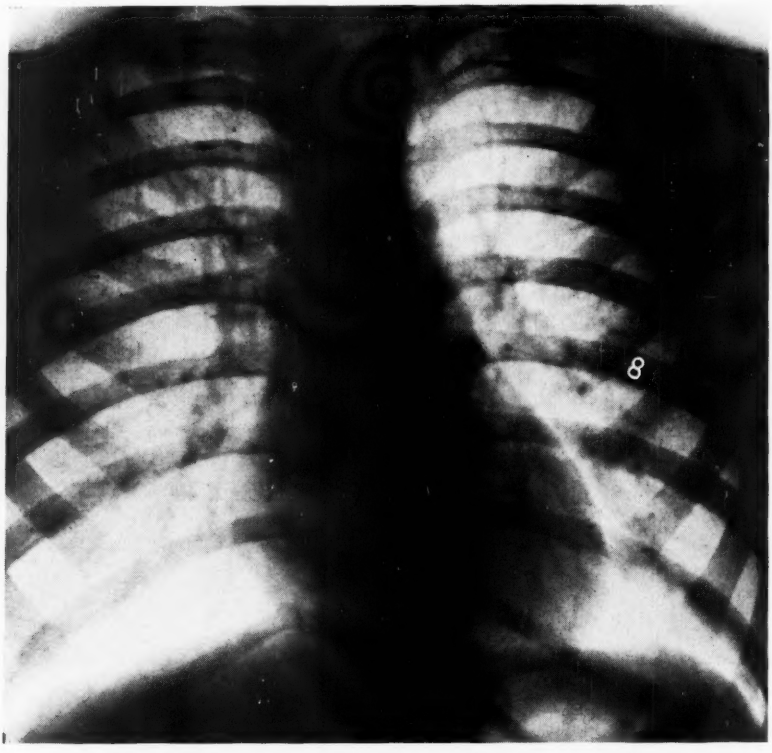


Fig. 2.

wedged in so tightly that no proper forceps place could be found, and all attempts at extraction failed. Tracheotomy was performed the next day, after which the pellet was removed through the medium of bronchoscopy, with great difficulty. The diameter of the pellet was 6.5 mm., certainly very large for this part of the bronchus of the lower lobe in such a young child. Extraction was followed by a speedy recovery.

Bronchography was made after a few days, and 10 c.c. of lipiodol were introduced; as appears from Fig. 4, the bronchial tree was entirely normal. The bronchogram was important, because it told us more about the exact localization of the foreign body. Figs. 2 and 3 show that the pellet remained practically in the same place, in spite of deep respiration. During expiration there was a slight lateral movement, but on both photographs it is on a level with the eighth rib. It is a well-known fact that, whereas there is a considerable dislocation in the distal parts of the lung during deep respiration, there is only little movement near the hilus and the great bronchi.

Returning to the eighth rib on the bronchogram of Fig. 4, we find that the pellet was localized in the entrance of the bronchus of the lower lobe of the left lung, but far enough down to leave the passage to the great first dorsal side branch free. This first dorsal branch is very large and always present. During the last few years it has been examined by several investigators who described the anatomy of the bronchial tree. We, too, were able to confirm these results.[3] This side bronchus branches off a little higher than the former location of the pellet, almost opposite the fork of the bronchus of the upper lobe.

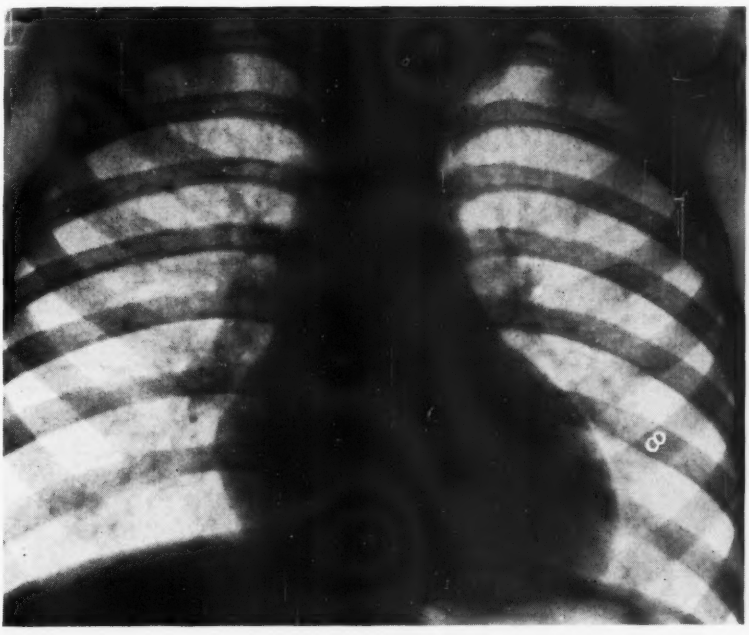


Fig. 3.

Fig. 5 gives a schematic representation of the situation in these two cases. In the first case (Fig. 5, *A*) there was a complete obstruction of the bronchus of the lower lobe, no collateral ventilation and, therefore, atelectasis. In the second case (Fig. 5, *B*) the first dorsal branch was free, and the only possible explanation seemed to be that there had here been collateral ventilation, as indicated by the arrows, so that no atelectasis had developed. It is a curious fact that no symptoms of bronchostenosis could be found. Investigations by Baarsma and Dirken showed that there was so much collateral ventilation in rabbits that this may explain why a considerable new supply of air is possible in man as well, and why there is no dislocation of the mediastinum during respiration. These cases, to which many more from the clinic might have been easily added, rendered the possibility of collateral ventilation in man very likely. In order to make sure, a more direct method to obtain confirmation was necessary.

METHOD

Evidently, only an entirely harmless method could be used when investigating collateral ventilation in human beings. It may even be asked if such experiments were permissible at all. In view of the importance of this problem, we answered this question in the affirmative, especially because the experiments involved no danger to the patient. Investigations have

only been made in cases where bronchoscopy was required for other reasons and where the lungs previously had been proved to be perfectly normal. The first patient was an asthmatic who was sent to our clinic for bronchoscopic treatment. We were greatly relieved that shortly after the departure of this patient, another from the same village was admitted, who brought a letter from the local physician to the effect that the first patient had been so pleased with the success of his careful treatment that the second wanted to be treated in the same way. The psychologic factor is of considerable importance with asthma. We have never, however, seen any harmful effects from these investigations.

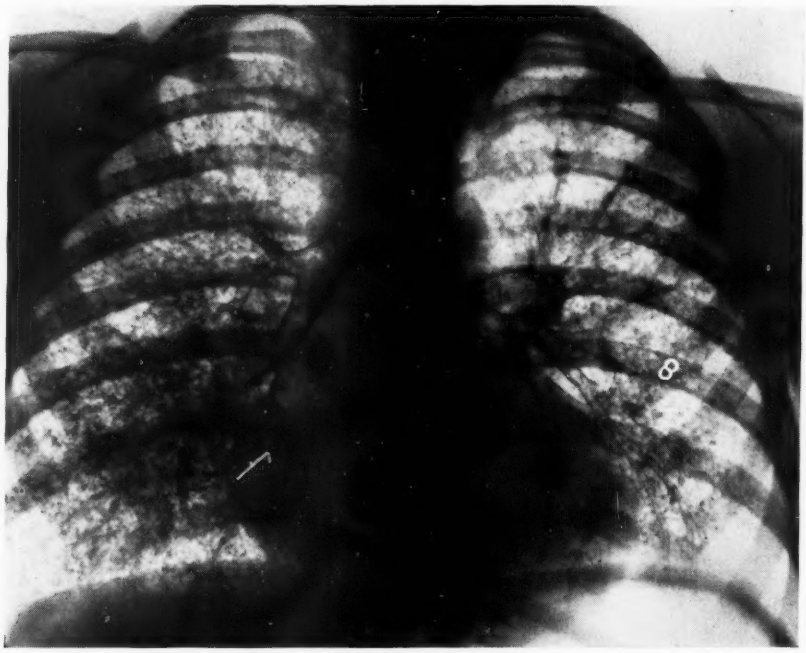


Fig. 4.

In obstructing the entrance to the bronchus, it was naturally impossible to make use of the laminaria cylinder described in a previous publication on experiments with rabbits. We used an inflated catheter, as used by Coryllos and others. It had a small rubber cuff attached to its distal end, which could be inflated. It was filled with a sterile salt solution by means of a very thin rubber tube running through the catheter. When the catheter was well in position and the rubber cuff inflated, the latter hermetically closed the entrance to the bronchus. The catheter could not be withdrawn unless the cut was first emptied, and it moved synchronously with respiration.

The patient was anesthetized by means of pantocain 2 per cent adrenalin, and put on the bronchoscopic table. The bronchoscope (after Jackson) was introduced, and the bronchus into which the catheter was to be inserted was adjusted. The lower lobe was used in all cases; in a number of experiments the catheter was inserted in such a way that the entire bronchus of the lower lobe was obstructed, in other cases only part of a lobe was disconnected by inserting the catheter into the posterobasal or laterobasal side branch of the bronchus of the lower lobe.

The catheter was then connected with the spirometer, which recorded the respiratory movements on a kymograph. A small weight caused a certain negative pressure in the spirometric system, and consequently in the part of the lung connected with it. If, therefore, there were collateral ventilation, air from the surrounding parts of the lung would be sucked into the area of negative pressure and the volume of the spirometer would be increased. This was recorded by a decline of the curve on the kymograph. If there were no collateral ventilation, the volume of the spirometer would remain more or less stationary, and consequently the curve on the kymograph would show a horizontal course. The whole experimental arrangement is shown in Fig. 6.

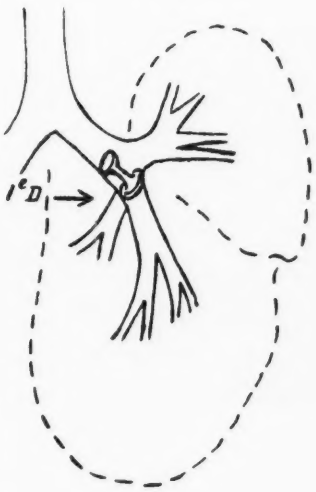


Fig. 5A.

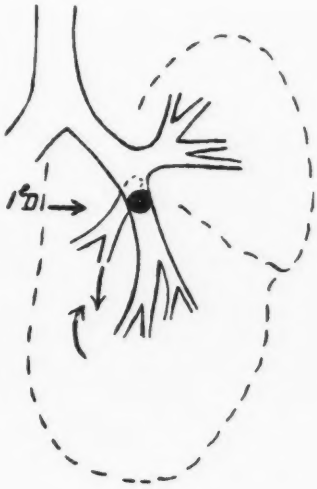


Fig. 5B.

When the whole of the lower lobe was disconnected, there was not collateral ventilation, as might be expected. Fig. 7 gives an instance of such a curve; the left lower lobe of a man of 28 years was disconnected completely. Respiration was recorded with a negative pressure of 0 to 2 cm. water in the disconnected lobe. The curve kept a horizontal course.

Fig. 8 illustrates the experiment with a man 26 years old, who had the catheter in the laterobasal section of the left lower lobe. The fall in the curve was very obvious here and was proportional to the negative pressure which was determined by the weight attached to the spirometer. It followed that collateral ventilation had been proved here. This was also the case in Fig. 9, the curve of a 31-year-old man, where the catheter had been inserted into the posterobasal branch of the left lower lobe. The volume of breath was much smaller here, and the decline of the curve was less. Yet there obviously was a decline of the curve, and consequently an increase of the volume of the spirometer, which increased with the negative pressure.

The same result was obtained on all occasions; there was no fall of the curve when the whole of the lobe was disconnected, but there was a fall when only a side branch was obstructed. *This proves with certainty that there is collateral ventilation in man.* The mechanism can hardly be imagined otherwise than in dogs and rabbits.

DISCUSSION

No doubt, collateral ventilation is highly important, first of all in physiology, as has been described in the preceding publication. It provides an equal

distribution of air and the speedy neutralization of even small pressure differences between various adjacent parts of the lung. This collateral ventilation is, however, of far greater importance in pathology. No doubt atelectasis would develop much more frequently without it.

It seems that in classifying atelectasis, the old way is still the best, and that in *acquired* atelectasis we can only distinguish *resorption* and *compression* atelectasis. The latter, which is caused by a passive emptying of the lung, as in the case of an exudate in the pleural cavity or a large tumor, need not be considered further.

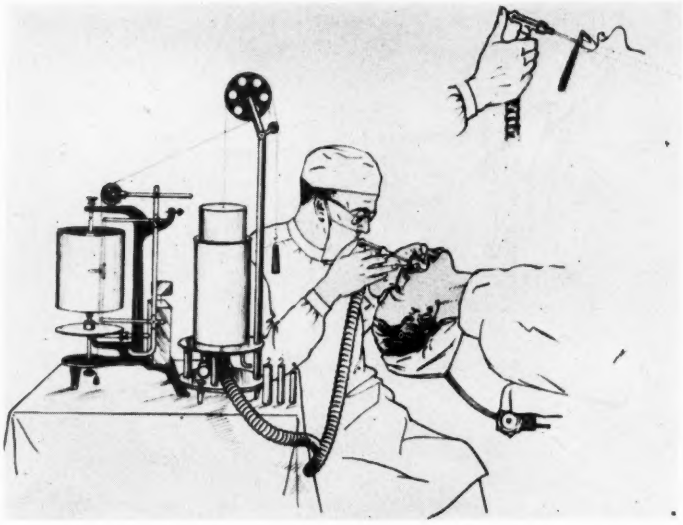


Fig. 6.

Resorption atelectasis develops because, as a result of an obstruction of the bronchus the air disappears from the part of the lung belonging to it. The remaining air is resorbed by the blood in the alveolar capillaries. Notably Coryllos and Birnbaum[4] have made extensive investigations as to how this resorption is effected. Complete obstruction of a main bronchus or a lobar bronchus causes atelectasis; obstruction of a side branch does this only under particular circumstances. We can now state that on the whole these circumstances are determined by the possibility of or absence of collateral ventilation under the given anatomic conditions, or by elimination of collateral ventilation under certain circumstances.

As to the first, it sometimes happens that a certain side branch provides for a segment of the lung which is completely separated from the remainder of the lobe. In our anatomic investigations, for instance, we found that in one out of every ten cases, the first dorsal segment of the lower lobe was defined by a more or less deep fissure. Like other investigators, we found that in a few cases this segment made the impression of a separate lobe (lobe moyen dorsal). Under

these circumstances, obstruction of this branch will automatically bring about atelectasis. We see, therefore, that anatomic variations are of considerable importance in this respect.

Collateral ventilation is eliminated (1) by shallow breathing, which minimizes pressure differences between various parts of the lung, or (2) it is eliminated if an inflammation develops in a part of the lung which is not disconnected.

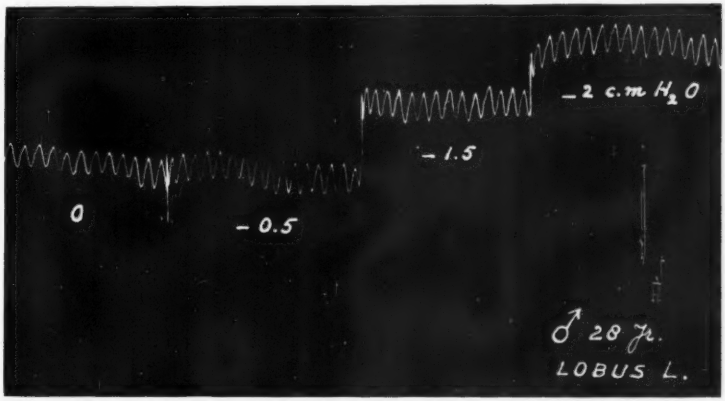


Fig. 7.

The latter could be proved experimentally by van Allen and his collaborators. The two factors will often go together; many patients with shallow breathing have at least a bronchitis. Each inflammatory process in the lung may easily cause obstruction of vents through edema or the presence of fluid or secretion, and consequently impede collateral ventilation. This factor of inflammation probably will be of the greatest consequence in most cases.

Shallow breathing may result from narcosis, or, if breathing is impeded, by pain or paralysis of the respiratory muscles. A great deal has been written during the last few years about postoperative collapse of the lung and traumatic atelectasis, that is, with injuries of the thorax. The mechanism has become much more intelligible, now that we have become acquainted with collateral ventilation. These patients are often suffering from bronchitis; plugs of secretion obstruct the bronchi and when their breathing becomes shallow as a result of pain, collateral ventilation is eliminated and atelectasis arises.

In patients with bronchitis, the presence of collateral ventilation often prevents atelectasis. If, for instance, a side branch of the bronchus is obstructed by a plug of secretion, a negative pressure arises behind the obstruction, which causes collateral ventilation. With a strong negative pressure, the plug will be sucked more firmly into the bronchus. Collateral ventilation, however, keeps down negative pressure and consequently there remains sufficient air behind the obstruction. Thanks to this air, the plug may be removed by coughing. This example clearly shows the importance of collateral ventilation in pro-

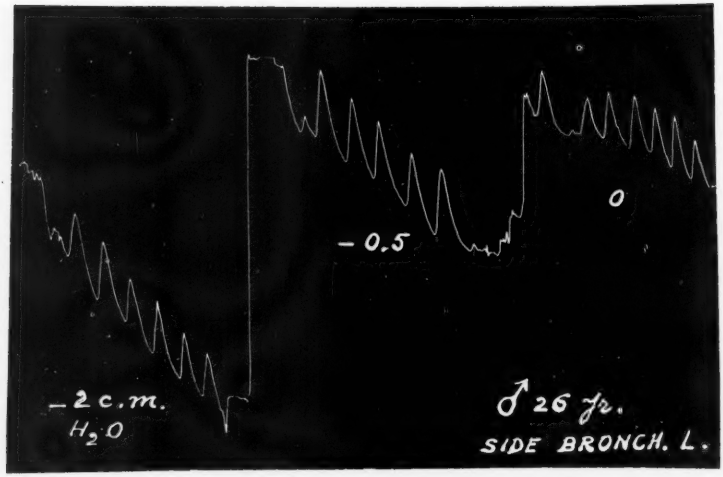


Fig. 8.

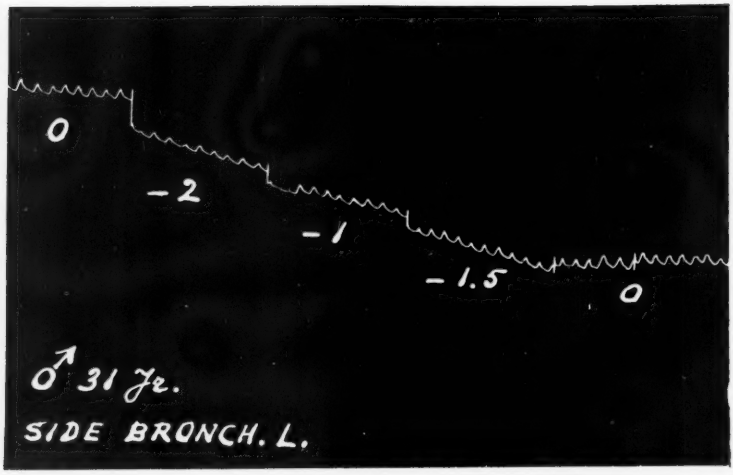


Fig. 9.

moting expectoration and preventing atelectasis, with much inherent mischief. For it is now known that there is only a very gradual transition from atelectasis to pneumonia.

The importance of collateral ventilation is also apparent with stripe-form atelectasis as described by Fleischner[5] and Keyser.[6] It has long been known that patients with widely divergent histories have shown stripe-form shadows on the radiogram, especially on the base of the lung. This was found in: patients with fractures of the ribs; kyphoscoliosis; after operations, especially of the higher abdomen; during coma, etc. Pleurisy was occasionally thought of, Which was supposed sometimes to develop lymphogenously from peritonitis.

Fig. 10.

Fleischner has been first to give the correct explanation. All these patients have shallow breathing and bronchitis in common. Obstruction of a bronchus causes the collapse of part of a lobe. The thoracic wall and the part nearest the hilus remain in their places. This part of the lung can only contract in a downward direction, and an atelectatic disk develops, which may be seen in the radiogram as a stripe. He called this "gerichteter Kollaps." Keyser was able to confirm these observations. Fig. 10 shows such a stripe-form shadow in a patient with multiple costal fractures. It is now clear that the essential factor in this

stripe-form atelectasis is the absence of collateral ventilation through shallow breathing of these bronchitic patients. These shadows disappear when a patient is made to breathe deeply.

It is even more important to prevent atelectasis, especially in view of a possible inflammation of the lungs. In this respect, the prophylaxis has been the common property of each practitioner for many years. He knows very well that deep breathing, the stimulation of expectoration, sitting up in bed, and getting up quickly, especially with older people, prevent a hypostatic pneumonia. Unconsciously, he has thus promoted collateral ventilation. Old experiences have now been given a scientific coloring. It is valuable if the right theoretical explanation can be given for practical facts.

With massive atelectasis of the whole lung or lobe, there is only one important factor, the obstruction of the main bronchus or the bronchus for a lobe. With the very frequent focal atelectasis, there are, consequently, two factors: the obstruction of a side branch and the elimination of collateral ventilation. This does not apply to a specific inflammation only, but also to tuberculosis; Westermark[7] and Roth[8] have demonstrated how easily bronchi in a tuberculous area may be obstructed by caseous masses, coagulation of blood, granulations, and secretions, which repeatedly cause atelectasis. These findings, as well as the stripe-form atelectasis of Fleischner, have been confirmed by dissection. But all this cannot be brought about, except by the elimination of collateral ventilation.

It is possible that this elimination of collateral ventilation is also connected with the *selective collapse of the lung.* It would take us too far afield to deal with the whole question of atelectasis. We will mention only the fact that French and Italian authors such as Cardis,[9] Chadourne,[10] and Parodi[11] have drawn attention to the so-called *reflective collapse of the lung.* Through the action of the pulmonary musculature, which has been the object of much controversy recently, a lobe or a whole lung is supposed to collapse. This explains syndromes such as postoperative and traumatic collapse of the lung. Selective collapse of the lung is observed particularly in the case of a pneumothorax induced in the treatment of pulmonary tuberculosis. In these cases, the affected parts of the lung often collapse to a greater extent than the nonaffected ones. Good illustrations of this phenomenon may be found in a report by Bronkhorst and Dijkstra.[12] It is explained by different tonic conditions in the diseased parts of the lung. It may also be imagined, however, that certain side branches in the affected part of the lung are obstructed. The pneumothorax reduces the ventilation in that lung, the collateral ventilation is eliminated, and an atelectasis develops. In any case the mechanism of obstructing one or more side branches and eliminating collateral ventilation must be taken into account in explaining the phenomenon of atelectasis.

RECAPITULATION

After collateral ventilation had been established in dogs (van Allen and collaborators) and rabbits (Baarsma and Dirken), it now has been proved to exist in man as well. An entirely harmless method of recording the phenomenon

in man is described. When the bronchus for a lobe is obstructed there is no collateral ventilation, and atelectasis follows. When a side branch is obstructed, there is no atelectasis, due to collateral ventilation. Atelectasis develops only if there is no collateral ventilation due to certain anatomic conditions, or if it is eliminated as a result of an inflammation or shallow breathing. The great importance of collateral ventilation for the clinic is further described.

REFERENCES

1. Baarsma, P. R., and Dirken, M. N. J.: J. THORACIC SURG. **17**: 238, 1948.
2. Van Allen, C., and Lindskog, G.: Arch. Surg. **21**: 1195, 1930.
   Van Allen, C., and Jung, T. S.: J. THORACIC SURG. **1**: 1, 1932.
3. Behr, E., Huizinga, E., and Pothoven, W. J.: Acta Radiol. **19**: 399, 1938; **21**: 314, 1940; **24**: 226, 1943.
4. Coryllos, P. N., and Birnbaum, G. L.: Arch. Surg. **16**: 501, 1928; **18**: 15, 1929.
5. Fleischner, F.: Fortschr. Röntgenstr. **53**: 607, 1937.
6. Keyser, S.: Nederl. Tijdschr. Geneesk. 6084, 1937.
7. Westermark, N.: Acta Radiol. (Stockh.) **16**: 531, 1935.
8. Roth, P.: Atelektasen bei kavernoser Lungentuberkulose, Leipzig, 1937.
9. Cardis, F.: Bull. Soc. méd. Hôp. Paris, 259, 1936.
10. Chadourne, P.: Diss. Paris, 1936.
11. Parodi, F.: Rev. de la Tuberc. **2**: 1231, 1936.
12. Bronkhorst, W., and Dijkstra, C.: Beitr. klin. Tbk. **94**: 445, 1940.

STUDIES ON EXPERIMENTAL PHOSGENE POISONING

II. Pulmonary Artery Pressure in Phosgene-Poisoned Cats

Mary H. Gibbon, H. D. Bruner, M.D., Ph.D., Robert D. Boche, Ph.D., and John S. Lockwood, M.D.
Philadelphia, Pa.

IN SPITE of extensive study following the use of phosgene in chemical warfare, the mechanism by which this agent produces pulmonary edema is still obscure. Two general theories have been proposed: According to one, the edema is attributed to direct (toxic) injury to the endothelium of the pulmonary capillaries, while according to the other, the hydrostatic pressure within these vessels is believed to rise to levels sufficient to overbalance the colloidal osmotic pressure of the plasma. A third possibility, obstruction to the lymphatic drainage from the lung, has not received serious consideration except for recognition that the lymphatics seem inadequate to drain away significant amounts of alveolar fluid.[1-4]

The more probable concept of toxic endothelial damage has not been conclusively established, because efforts to demonstrate histologic evidence of endothelial injury have failed. Consequently, the alternative explanation has persisted, although its supporting evidence is indirect and unsatisfactory. This evidence consists of the reputed association of "blue" or "plum-colored" cyanosis (implying venous congestion) with phosgene poisoning in man,[5] and the occurrence of multiple thromboses or other changes assumed to be obstructive, in the pulmonary vascular system.[6-8] A modification of this general concept is Rothlin's[9] suggestion that the edema is brought about by a reflexly mediated vascular response but, since the experimental evidence on which this proposal was based could not be confirmed,[10] it may be dismissed from further consideration.

Arguments against the elevated pressure hypothesis may be summarized as follows: (a) The edema fluid, histologically and by analysis, is nearly identical with the blood plasma;[4, 10, 11] it is not a protein-poor filtrate of plasma. (b) There is reason to be skeptical of statements that the "blue" cyanosis and venous distention are a consequence of phosgene poisoning.[12] So far as can be determined from the literature, "blue" cyanosis has been reported only in field casualties of World War I, and in neither accidental phosgene poisonings[13-19] nor in experimentally poisoned animals.[8, 11, 20-22] It is clear from the literature of 1915 that chlorine does cause "blue" cyanosis and the subsequent use of mixtures of gases[5, 6, 14, 20] encourages the view that the "blue" cyanosis

From the Harrison Department of Surgical Research Schools of Medicine, University of Pennsylvania, Philadelphia.

The work described in this paper was done under a contract recommended by the Committee on Medical Research, between the Office of Scientific Research and Development and the University of Pennsylvania.

Received for publication May 16, 1947.

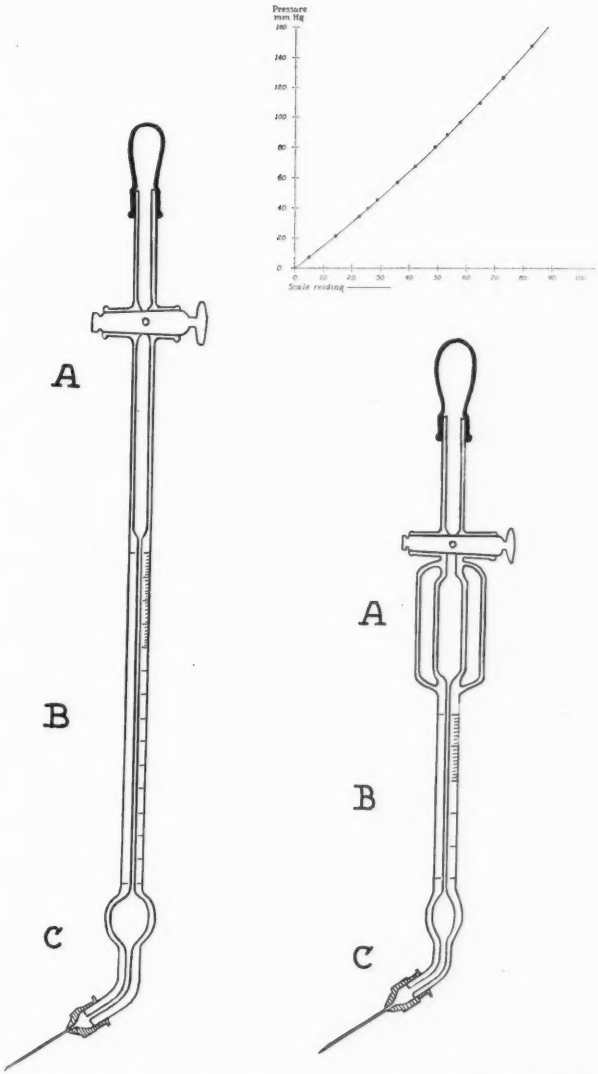


Fig. 1.—Line drawing of manometers used for measuring systemic (left) and pulmonary artery (right) blood pressures. The insert is a calibration curve for the systemic manometer.

of reputed phosgene casualties really was observed in mixed gas poisoning. (c) As reported elsewhere,[10] thromboses are exceptional in animals dying spontaneously within forty-eight hours after exposure to median concentrations of pure phosgene; the earlier reports of thrombosis seem not to have been interpreted with regard to the probable conditions of exposure. (d) Dogs and rats which were thoroughly heparinized before and continuously after exposure to phosgene have developed typical edema.[21] (e) Measurements of right ventricular pressure in phosgene-poisoned goats and dogs[22, 23] lead to the inference that pulmonary artery pressures are not abnormal. (f) Daly and

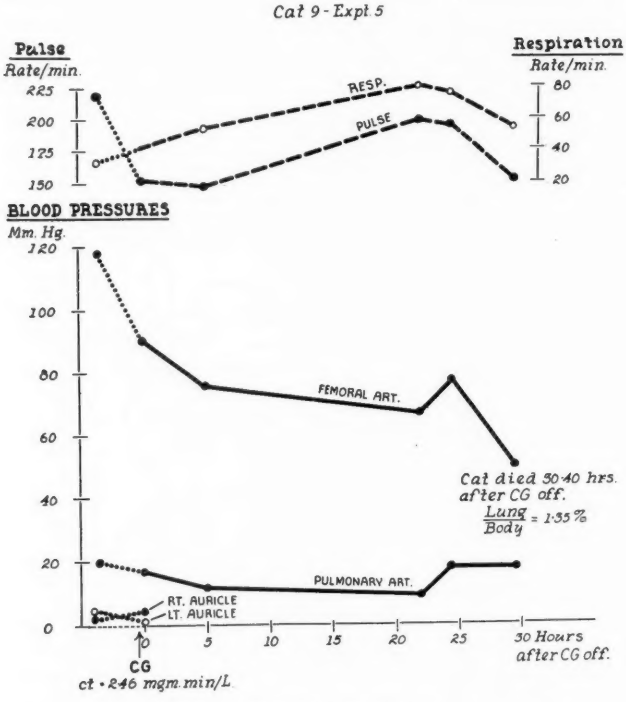


Fig. 2.—Phosgene poisoned cat.

associates[24] have observed no appreciable rises in pulmonary artery pressures in perfused dog lungs until nonspecific edema was present in both the gased and the control lung; their evidence suggests vasodilatation and pooling in the isolated gassed lung.

These considerations, however, do not eliminate the possibility of elevated pulmonary vascular pressures as a factor in the pulmonary edema produced by phosgene. In view of its basic importance, the problem was attacked by direct measurement of the pulmonary artery pressure and by evaluation of the pulmonary pressure gradient.

METHODS

Cats were used in this study, because their thoracic structure lends itself best to a marsupialized heart preparation which permits access to the heart and great vessels in the unanesthetized animal.[25] At a preliminary operation, the left fourth and fifth costal cartilages with bits of the adjacent ribs were resected, the pericardium sutured securely to the margins of the defect, and the

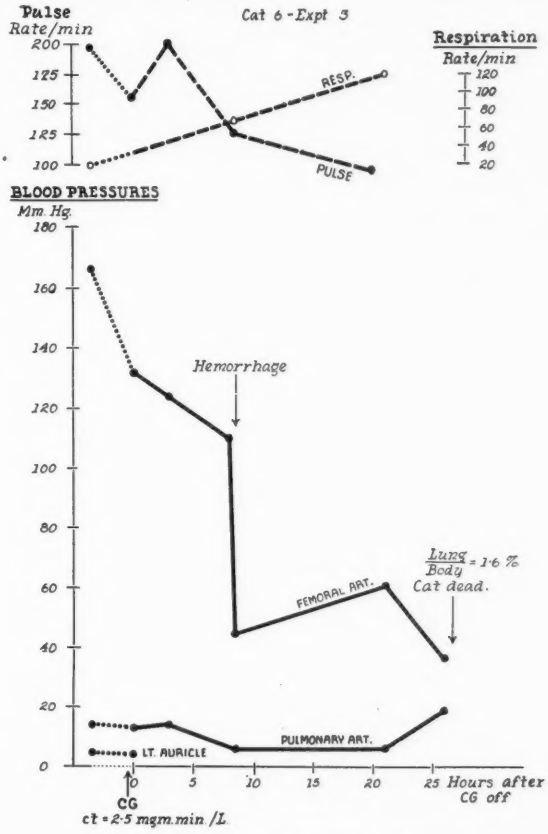


Fig 3.—Phosgene poisoned cat.

wound closed. After complete recovery in three to five weeks, the skin and pericardium covering the opening were incised under very light anesthesia (Pentothal Sodium or Evipal) a few hours before gassing; the femoral artery also was exposed. Normal cats were similarly anesthetized and the femoral arteries isolated; these animals served also as gas chamber controls.

The prepared animals, each paired with a control, were exposed to varying doses of phosgene in a dynamic chamber by the standard "push-pull" technique described elsewhere.[10] The concentration-time (CT) products

of exposure, in milligram-minutes per liter, were selected to produce varying degrees of severity of poisoning and edema; the mean concentration (C) was 0.29 mg. per liter ± 7 per cent, the duration of exposure (T) being varied from 8½ to 13 minutes.

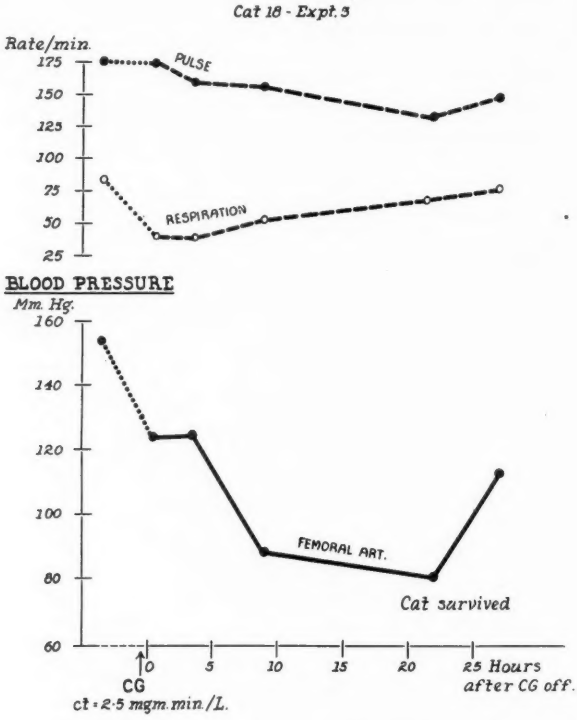


Fig. 4.—Phosgene poisoned cat (a simultaneously gassed control).

At death or sacrifice, complete autopsies were performed and the organs studied histologically. In the animals living longer than twenty-four hours, a fibrinopurulent pericarditis developed which was only partially controlled by sulfonamides.

At intervals after gassing, the animals were restrained in the supine position and the pressures were measured by means of manometers designed and built by R. D. Boche (Fig. 1). In operation, isotonic citrate is drawn into the bulb (C) to the zero mark on the calibrated stem (B); with the manometer vertical the stopcock is then closed, and the needle inserted into the vessel, taking care to keep the manometer vertical and to avoid warming the air-containing portion (A). Theoretical and experimental calibrations of the stem agreed almost perfectly. The recorded values represent mean pressures due to the damping characteristics of the instruments. Since the needle on the manometer was directed centrally in the vessels, the recorded pressures are the sum of the velocity and static pressure heads.

Because each reading necessitated puncture of the vessel or heart wall, the number of readings was kept to the minimum necessary for the required information. Between readings the cats were unrestrained.

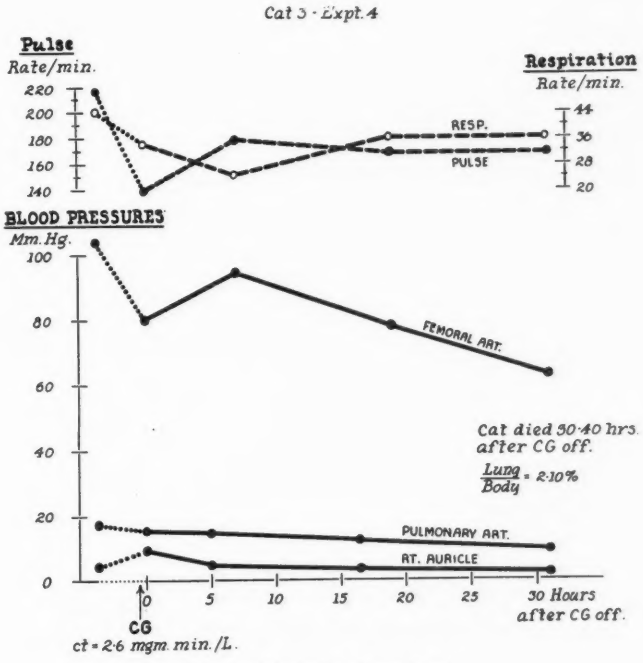


Fig. 5.—Phosgene poisoned cat.

RESULTS

The data on five prepared cats and on two of the five simultaneous controls are presented in graphic form in Figs. 2 to 8 (Figs. 4 and 8 are control animals) in order of increasingly severe exposure to phosgene (CG). The signs and course of poisoning in the prepared animals were in all respects similar to those in the controls. The inadvertent hemorrhage noted in Fig. 3 consists of a 10 to 15 c.c. loss from the femoral artery.

It is evident that the pulmonary artery pressures rarely rose above the pregassing values except terminally; the tendency was toward a fall, synchronous with the systemic pressures. With one exception, the animals showed the typical bradycardia at once after exposure, but thereafter the pulse and respiratory rates varied widely, attributable in part to the degree of acquiescence by the animal to the supine position. The majority of the readings were taken after the animal had relaxed, but in a few instances the value noted is that obtained between bouts of struggling or meowing. Such values may be some-

what higher than those which would have been obtained otherwise. The supine position seemed especially objectionable to the cats after edema had begun to develop.

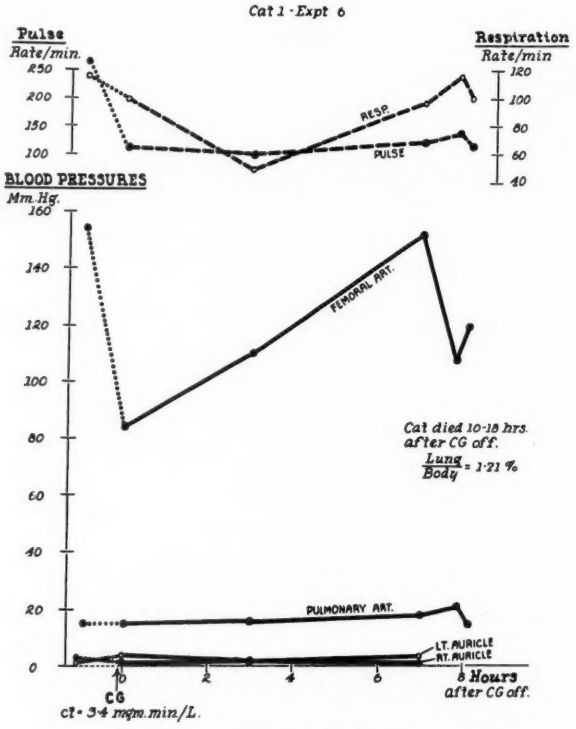


Fig. 6.—Phosgene poisoned cat.

Although few in number, measurements of pressure in the right and left auricles indicate that no essential changes were present after gassing.

Judged both by gross and histologic examination and by increase of the lung/body weight ratio, these cats showed a lesser degree of pulmonary edema in proportion to their clinical condition than was usually found in dogs. Hemoconcentration also was less pronounced in cats during the course of poisoning and at death.

DISCUSSION

The pulmonary artery pressures before exposure to phosgene lie within the range of pressures observed in similarly prepared cats using a different type of manometer,[26] and recorded values for mean pulmonary artery pressure in cats and in other species by various techniques are comparable with those recorded here.[27-29] While exposure of the intrapericardial structures to atmospheric pressure undoubtedly results in the development of abnormal pressure relations,[30] the validity of these data is not affected by these experimental conditions, since (a) the pulmonary artery pressures before gassing

were normal, and (b) they fell after gassing instead of rising, as would have occurred if the preparation had predisposed *per se* toward appreciable accumulation of blood in the lungs. Furthermore, the chief interest centers on the relative pressures before and after gassing. The slight terminal rises of pressure in Figs. 2, 3, 6, and 7 may be due in part to the accentuated expiratory efforts characteristic of advanced phosgene poisoning.[11]

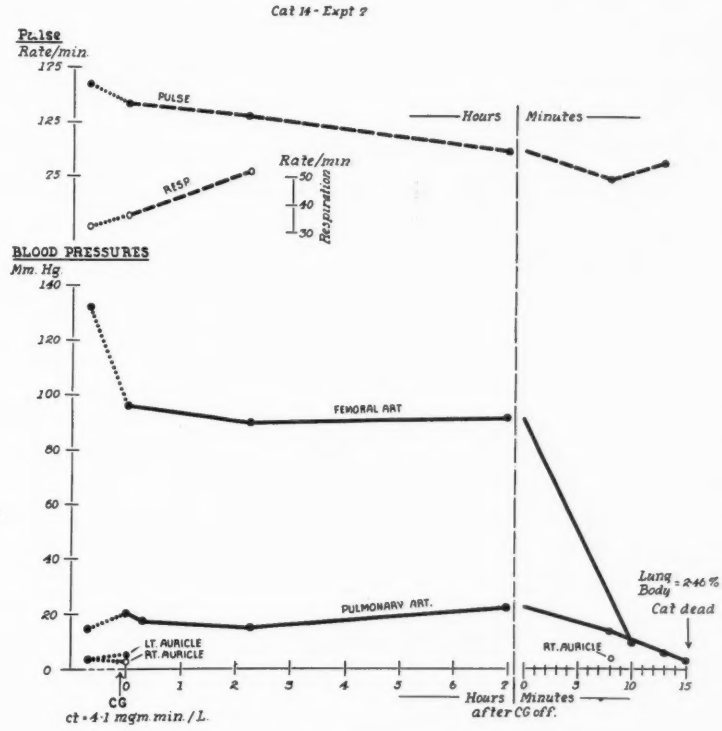


Fig. 7.—Phosgene poisoned cat.

The observations of left auricular pressure, although unavoidably incomplete, support the assumption that the pressure gradient across the lung follows the usual slope. There is no anatomic evidence to suggest that the major pressure loss occurs distal to the pulmonary capillaries. There seems to have occurred an effective adjustment by the lesser circulation toward maintenance of nearly normal pressure levels and gradient, in the presence of extensive disturbances of systemic pressure, heart rate, blood volume and viscosity, and the development of severe or lethal edema.

These data, together with the evidence outlined in the introduction, make untenable the hypothesis that the pulmonary edema of phosgene poisoning is of purely hydrostatic origin. This statement is further strengthened by the fact that the preparation employed should have exaggerated the hydrostatic

factors leading to edema. It is obvious, however, that maintenance of any pressure sufficient to sustain circulation contributes to continued outflow of fluid into the alveoli, whatever the primary mechanism initiating the edema.

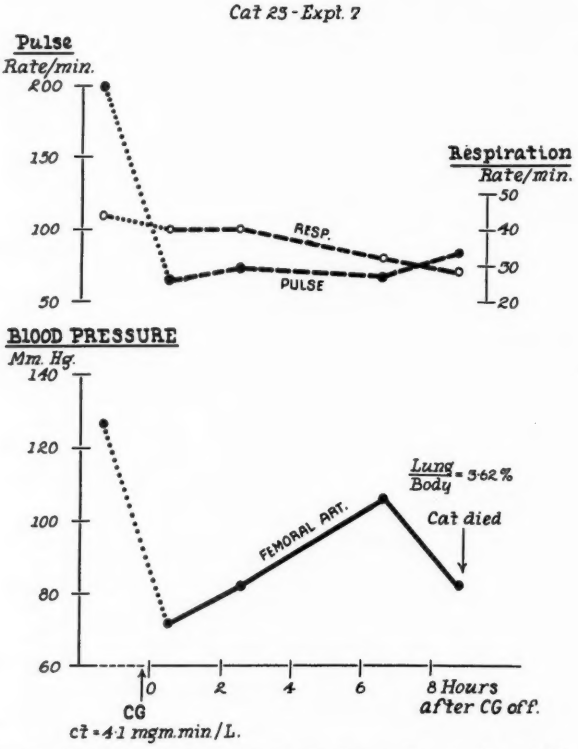


Fig. 8.—Phosgene poisoned cat (a simultaneously gassed control).

Since there appear to be no reasons for assuming that the cat's anatomic and physiologic response to phosgene is peculiar to that species in any fundamental respect, these data lead to the following suggestions regarding the therapy of phosgene poisoning in man: (1) There appears to be no indication for venesection, which previously has been carried out for the purpose of reducing pulmonary and systemic venous congestion. (2) Drugs tending to increase pulmonary artery pressure, e.g., Adrenalin,[27] should not be given. (3) Because the observed hemodynamics may represent an effective body adjustment to phosgene damage, it may be unwise to interfere by means of drugs or procedures intended to support or depress a particular sector of the circulation or heart. The most pressing defect is anoxic anoxia and attention should be concentrated upon relieving it.

SUMMARY

1. Pulmonary artery, and right and left auricular pressures have been measured directly in five cats poisoned with phosgene.

2. Abnormal elevations of pressure were not found after gassing.

3. Collateral evidence is cited for dismissing the hypothesis of an hydrostatic origin of pulmonary edema in phosgene poisoning.

REFERENCES

1. Drinker, C. K., and Warren, M. F.: The Genesis and Resolution of Pulmonary Transudates and Exudates, J. A. M. A. **122**: 269, 1943.
2. Miller, W. S.: The Lung, Springfield, Ill., 1936, Charles C Thomas.
3. Chemical Warfare Medical Committee, Report No. 2 by Edkins, J. S., and Tweedy, N.: The Early Changes Produced in the Lungs by Gas Poisoning, and Their Significance, London, April, 1918, H. M. stat. off.
4. Cameron, G. R., and Courtice, F. C.: The Production and Removal of Oedema Fluid in the Lung after Exposure to Carbonyl Chloride (Phosgene), J. Physiol. **105**: 175, 1946.
5. Gilchrist, H. L.: The Medical Department of the United States Army in the World War, vol. 14, chap. 7, Symptoms and Treatment, pp. 250-271, Washington, 1926, Government Printing Office.
6. Winternitz, M. C., *et al.*: Collected Studies on the Pathology of War Gas Poisoning, New Haven, 1920, Yale University Press.
7. Dunn, J. S.: The Effects of Multiple Embolism of Pulmonary Arterioles, Quart. J. Med. **13**: 129, 1920.
8. Meek, W. J., and Eyster, J. A. E.: Experiments on the Pathological Physiology of Acute Phosgene Poisoning, Am. J. Physiol. **51**: 303, 1920.
9. Rothlin, E.: Pathogénie et thérapeutique de l'intoxication par le phosgène, Schweiz, med. Wchnschr. **71**: 1526, 1941.
10. Coman, D. L., Bruner, H. D., Horn, R. C., Jr., Friedman, M., Boche, R. D., McCarthy, M. D., Gibbon, M. H., and Schultz, J.: Studies on Experimental Phosgene Poisoning, with Special Reference to the Early and Late Phases, Am. J. Path. **23**: 1037, 1947.
11. Laqueur, E., and Magnus, R.: Über Kampfgasvergiftungen: III. Experimentelle Pathologie der Phosgenvergiftungen, Z. ges. exp. Med. **13**: 31, 1921.
12. Haldane, J. S.: Lung Irritant Gas Poisoning and Its Sequelae, J. Roy. Army med. Cps. **33**: 494, 1919.
13. Gerber, I.: Un cas d'intoxication mortelle par le phosgene, Rev. méd. Suisse rom. **40**: 356, 1920.
14. Hegler, C.: Ueber eine Massenvergiftung durch Phosgengas in Hamburg: I. Klinische Beobachtungen, Deutsche med. Wchnschr. **54**: 1551, 1928.
15. Jones, A. R.: The Treatment of Casualties from Lung Irritant Gases with Particular Reference to the Use of Oxygen and Carbon Dioxide Mixtures, J. Indust. Hyg. and Toxicol. **22**: 235, 1940.
16. Longcope, W. T., and Luetscher, J. A., Jr.: Personal Communication, 1944.
17. Roos, A.: Phosgenvergiftungen, Vjschr. gerichtl. Med. **48**: 67, 1914.
18. Steel, J. P.: Phosgene Poisoning; Report on Two Cases, Lancet **1**: 316, 1942.
19. O'connor, J. A.: Personal communication, 1943.
20. Underhill, F. P.: The Lethal War Gases, New Haven, 1920, Yale University Press.
21. Unpublished data.
22. Patt, H. M., Tobias, J. M., Swift, M. N., Postel, S., and Gerard, R. W.: Hemodynamics in Pulmonary Irritant Poisoning, Am. J. Physiol. **147**: 329, 1946.
23. Dunn, J. S.: Measurement of Pressure Changes in the Right Ventricle by Cardiac Puncture, Quart. J. Med. **13**: 46, 1919.
24. Daly, I. de B., Eggleton, B., Elsden, S. R., and Hebb, C. O.: A Biochemical Study of Isolated Perfused Lungs with Special Reference to the Effects of Phosgene, Quart. J. Exp. Physiol. **33**: 215, 1946.
25. Gibbon, J. H., Jr.: The Maintenance of Life During Experimental Occlusion of the Pulmonary Artery Followed by Survival, Surg., Gynec., & Obst. **69**: 602, 1939.
26. Gibbon, J. H., Jr., Gibbon, M. H., and Kraul, C. W.: Experimental Pulmonary Edema Following Lobectomy and Blood Transfusion, J. THORACIC SURG. **12**: 60, 1942.
27. Hamilton, W. F., Woodbury, R. A., and Vogt, E.: Differential Pressures in the Lesser Circulation of the Unanesthetized Dog, Am. J. Physiol. **125**: 130, 1939.
28. Katz, L. N., and Steinitz, F. S.: Pulmonary Arterial Pressure in Experimental Renal Hypertension, Am. J. Physiol. **128**: 433, 1940.
29. Czebrinski, E. W., Smith, J. R., Nemec, S. S., and Robb, J. A.: Measurement of Pulmonary Arterial Pressure in Dogs, J. Lab. & Clin. Med. **30**: 849, 1945.
30. Beck, C. S., and Cox, W. V.: The Effects of Pericardiostomy on the Mechanics of the Circulation, Arch. Surg. **21**: 1023, 1930.

THE LOCALIZATION OF PULMONARY CAVITIES BY A MODIFIED ROENTGENOGRAPHIC TECHNIQUE

JOSEPH GORDON, M.D.*
RAY BROOK, N. Y.

THE size and location of a tuberculous pulmonary cavity determines the extent of a thoracoplasty. The conventional posteroanterior roentgenogram ordinarily used is not always satisfactory for accurate localization of the cavity with relation to the overlying ribs. A more reliable method for determining its position will be described.

The technique for roentgen ray examination of the chest is not standardized. In communications from several members† of the Committee on X-Ray Apparatus and Technique of the American Trudeau Society, the tube position mentioned varied from the fourth vertebral space, sixth vertebral space to 6 inches from the top of the casette. However, practically everyone uses the posteroanterior projection. Since the primary purpose of the routine x-ray examination of the chest is the detection of abnormal shadows and not accurate localization, this technique is generally satisfactory. For more accurate study the central ray of the tube should pass through the object being examined and the projection should be such that the object is adjacent to the casette.

In the majority of instances antra are situated in the upper and posterior part of the lung. In this situation the anteroposterior projection is to be preferred for accuracy rather than the routine posteroanterior projection. Distortion is further diminished and accurate localization enhanced by placing the x-ray tube on the same level as the cavity, that is, centering the tube on the cavity. Both projection and centering are determined by means of fluoroscopic examination and then x-ray films are obtained. The discrepancies due to changing the tube position and projection were demonstrated by the following procedures:

An object simulating a cavity was placed within the thorax of a skeleton, its position fixed and noted. Fluoroscopic examination was then carried out and the varying location of the image on the screen observed. For example, with the cavity known to be at the level of the fourth rib posteriorly, the annular shadow appeared to be at the level of the fifth rib when the tube was placed at midthoracic level using the posteroanterior projection. The tube and screen were then centered on the cavity by reducing the screen aperture so as just to encompass the annular shadow. Here the shadow appeared at the level of the fourth rib. In the anteroposterior projection, with the tube at mid-thorax, the annular shadow appeared to be slightly above the fourth rib or somewhat higher than its known position. When the tube was "centered," so that the central ray

Read before the Inaugural Meeting of the Upstate Society of Thoracic Surgeons, Oct. 5, 1946, Albany, N. Y.
Received for publication June 9, 1947.
*Principal Thoracic Surgeon, Ray Brook State Tuberculosis Hospital.
†Personal communications.

passed through the cavity, the correct location was visualized. The difference here was that in the anteroposterior projection the object was nearer the screen and magnification of the cavity was less in contrast to the posteroanterior projection. For a cavity near the anterior chest wall, distortion in size was, of course, less in the posteroanterior projection. Films taken of the skeleton with the x-ray tube in positions similar to that of fluoroscopy showed the same variations in location of the annular shadow. (Fig. 1.)

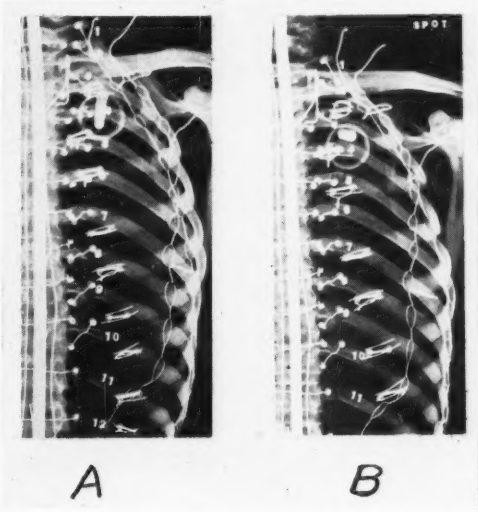


Fig. 1.—*A*, Posteroanterior roentgenogram of skeleton with simulated cavity located posteriorly and seen at fifth rib.

B, Anteroposterior spot roentgenogram showing cavity located in fourth interspace and known to be in this location.

By means of fluoroscopy and roentgenography, studies were made of patients with intrapulmonary cavities. The fluoroscopic findings corresponded with those noted above when using the skeleton. A series of films were then taken, conventional posteroanterior and anteroposterior, stereoscopic pairs, laterals, fluoroscopic spot films in both posteroanterior and anteroposterior projection, and "special spot" films, posteroanterior and anteroposterior. The fluoroscopic spot films were taken with the tube and screen centered as described above, the shutter being opened full for x-raying after fixing the position of the tube. The "special spot" films were obtained in the following manner: The annular shadow, localized by the same fluoroscopic method, was outlined on the skin both in front and back. The patient was then taken to the x-ray room and placed before the casette and the tube stand moved up to align the center of the x-ray tube with the skin mark. With the tube anchored at this level, the tube stand was moved back to a distance of 72 inches and films were taken in both anteroposterior and posteroanterior projections. Lateral films were taken at the same level at the recommended 48-inch distance.

The group of films taken on the same patient were then compared and from fluoroscopic studies and lateral films, the position of the cavity in relation to the anterior or posterior chest wall was shown. The cavity which lay nearer the posterior chest wall appeared lower in the conventional posteroanterior film and somewhat higher in the anteroposterior film than the level determined by fluoroscopy. In the films taken with the tube aligned to the skin markings ("special spot" technique), the location of the annular shadow in the anteroposterior pro-

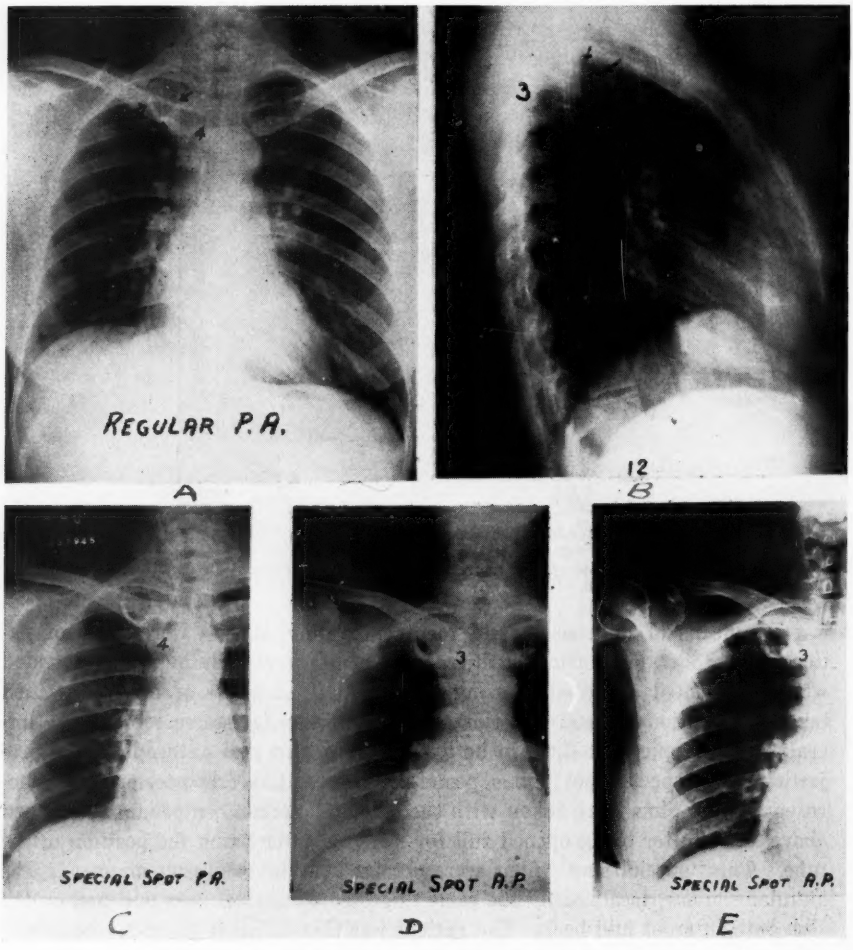


Fig. 2.—*A*, Conventional posteroanterior film showing annular shadow at level of fourth rib.

B, Right lateral film showing annular shadow at level of third rib and located well posteriorly.

C, Film taken with tube located at level of cavity but still using postercanterior projection. Annular shadow moved upward to third interspace above fourth rib.

D, Film taken as in *C*, but projection is anteroposterior for posterior cavity, which is now seen at third rib.

E, Film taken as in *D*, but with Bucky technique for sharper detail.

jection corresponded with the fluoroscopic centering and the posteroanterior showed only slight changes, chiefly those of magnification. The fluoroscopic spot films were similar to the special technique but magnification and distortion were present because of the short tube distance. The most striking difference was noted in comparing the conventional posteroanterior film with the "special spot" anteroposterior film. (Figs. 2 and 3.) The stereoscopic films were inaccurate because the fusion image was that of two different positions, low tube and high tube without regard to the location of the lesion.

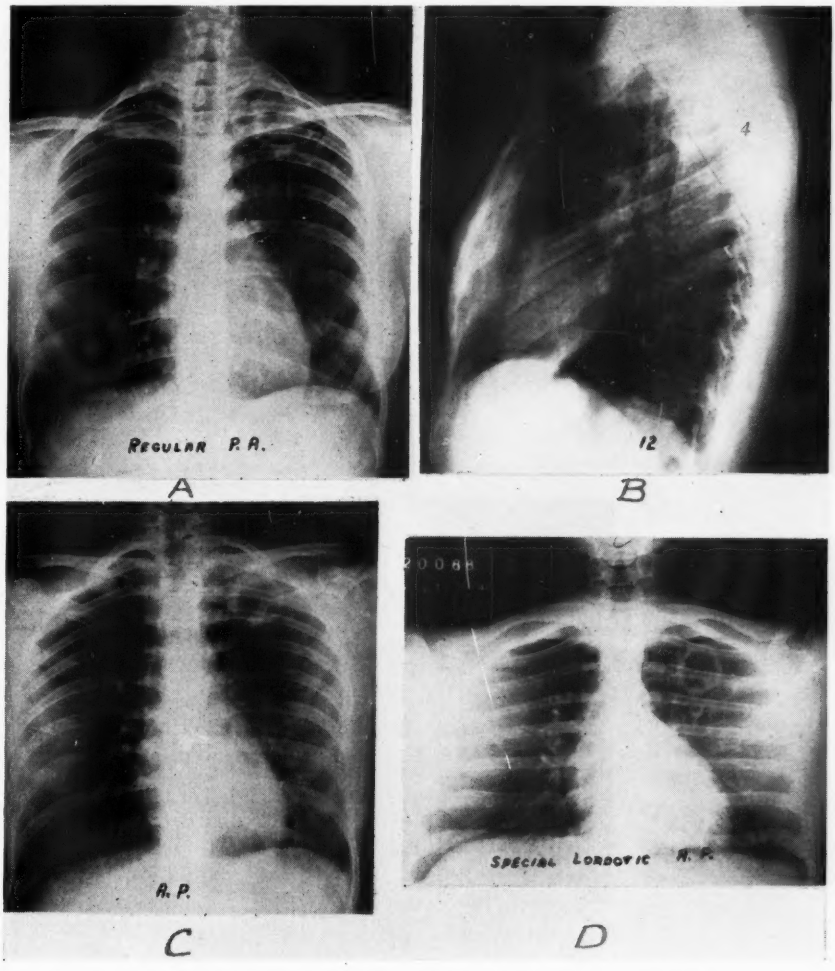


Fig. 3.—*A*, conven ional film showing annular shadow at level of fifth rib.

B, Left lateral film showing annular shadow at level of fourth rib and located posteriorly.

C, Film taken at level of cavity and correct or anteroposterior projection showing annular shadow at level of fourth rib.

D, Lordotic film taken in anteroposterior projection showing annular shadow above the plane of the fourth rib.

When the cavity lies virtually midway between the anterior and posterior chest walls, there is no significant difference in rib location in either the anteroposterior or posteroanterior spot films. Also, when the transverse diameter of the chest is increased because of kyphosis and the cavity thereby apparently lies about at the middle of this distance—no change in anteroposterior or posteroanterior spot is noted. When the cavity is in intimate proximity with the pos-

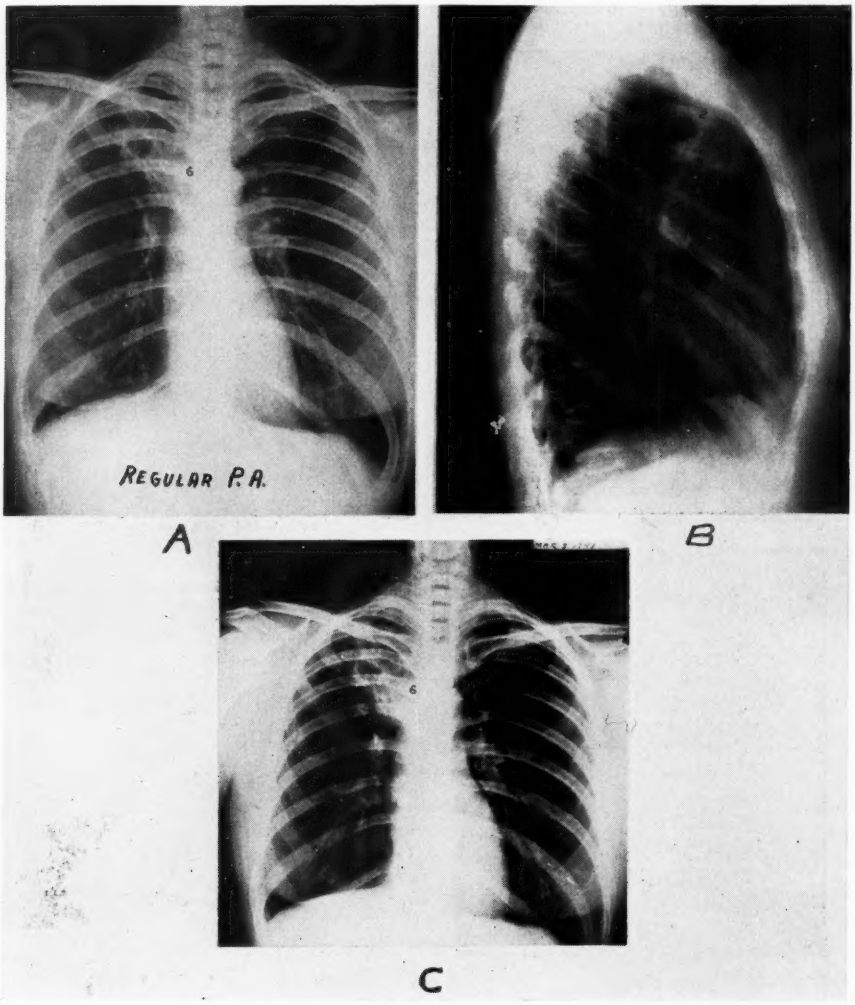


Fig. 4.—*A*, Regular film showing annular shadow at level of sixth rib.
B, Right lateral annular shadow at level of third rib but located well anteriorly.
C, Special spot film taken with tube at level of cavity and in posteroanterior projection because of anterior location of cavity. No essential difference is noted as the posteroanterior projection is correct for such a cavity and the sixth rib is midthoracic for tube location.

terior ribs, a regular anteroposterior film compared with a spot anteroposterior film shows insignificant change in location. The same is also true for a cavity in close proximity to the anterior chest wall when the films are taken in the posteroanterior projection. (Fig. 4.)

It would seem, therefore, that the relationship of a tuberculous pulmonary cavity to the overlying ribs is best determined by the technique indicated above. The proper projection is first determined by fluoroscopy or from a lateral film so that when films are taken, the position of the patient places the cavity the shorter distance from the casette. The alignment of the tube with the skin mark assures that the central ray passes through the object to be examined. The correction is in both the vertical and horizontal planes, thus considering the rib levels, the distance from the vertebral column, and the relationship to thoracic body lines.

In the technique of thoracoplasty operations it is generally advised that resection be carried one or two ribs below the cavity, assuming no significant lesions beneath it. On the basis of the above technique, it would be possible, in some cases, to resect fewer ribs and still be below the roentgen level of the cavity as contrasted with the shadow in the conventional film. Only a clinical trial, however, can determine the success attending the use of this type of roentgenogram as a guide to the extent of the operation.

Illustrations kindly prepared by Anna Sanlow, Senior X-ray Technician, Ray Brook, N. Y.

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

Twenty-Eighth Annual Meeting

May 31, June 1 and 2, 1948, Chateau Frontenac

Quebec

Preliminary Program

Monday Morning, May 31, 1948

9:00 A.M. BUSINESS MEETING

9:30 A.M. SCIENTIFIC SESSION

1. **Streptomycin as an Adjunct in Surgical Treatment of Pulmonary Tuberculosis**
   Daniel A. Mulvihill, Laurence Miscall and (by invitation) Robert Klopstock, Joseph Bitsack
2. **Role of Streptomycin in Surgery of Pulmonary Tuberculosis**
   P. T. Chapman (by invitation), E. J. O'Brien, Paul V. O'Rourke, and (by invitation) Bruce Douglas
3. **Experimental and Clinical Studies of the Role of Streptomycin in the Pleural Cavity**
   Edward J. Beattie (by invitation), Brian Blades, and (by invitation) Charles Horton
4. **Results in Pulmonary Resection for Tuberculosis (100 Cases Without Streptomycin Contrasted With 100 Cases Operated With Streptomycin Therapy)**
   Charles P. Bailey, and (by invitation) Thomas O'Neill, and Robert P. Glover
5. **An Evaluation of Streptomycin as a Protective Agent in Pulmonary Resections for Tuberculosis**
   J. A. Moore, J. D. Murphy, and (by invitation) P. D. Elrod
6. **An Anatomic Approach to Segmental Resection**
   J. Gordon Scannell (by invitation)
   Introduced by Edward D. Churchill

Monday Afternoon, May 31, 1948

2:00 P.M. SCIENTIFIC SESSION

7. **Endobronchial Occlusion During Pulmonary Resection**
   James D. Moody
8. **Medullary Pegging of Ribs in Thoracotomy Incisions**
   Karl P. Klassen
9. **Sputum in Cancer of the Lung: A Study of the Incidence and Causative Factors**
   John R. McDonald (by invitation), Stuart W. Harrington, and O. Theron Clagett
10. **Cytology of Bronchial Secretions—an Aid to Early Diagnosis of Lung Cancer**
    William L. Watson and (by invitation) Henry Cromwell, Lloyd Craver, and George N. Papanicolaou
11. **Malignant Nature of Bronchial Adenoma**
    Alfred Goldman
12. **Adenoma of the Bronchus**
    Harold Neuhof and (by invitation) Coleman B. Rabin

Tuesday Morning, June 1, 1948

9:00 A.M. SCIENTIFIC SESSION

13. **The Clinical Use of a Prosthesis to Prevent Overdistension of the Remaining Lung Following Pneumonectomy**
    Julian Johnson and (by invitation) Charles K. Kirby

14. **An Evaluation of Extrapleural Pneumonolysis With Lucite Plombage**
Josiah C. Trent (by invitation), James D. Moody, and Joseph S. Hiatt, Jr.
15. **Experimental and Clinical Study of Collapse Therapy Using Fiberglas Wool Fabric**
(By Invitation) Thomas J. O'Neill, Hector P. Redondo, and Robert G. Trout
16. **Intrathoracic Meningocele**
Francis X. Byron and (by invitation) Emery E. Alling, and Paul C. Samson
17. **Use of Pedicle Tube Flap in Carcinoma of Upper Esophagus**
Eugene Bricker (by invitation) and Thomas H. Burford
18. **Transthoracic Esophago-Jejunogastrostomy for High Stricture of the Esophagus**
Albert Wilson Harrison
19. **Completing the Multiple-Stage Operation for Atresia of the Esophagus**
Conrad R. Lam

Tuesday Afternoon, June 1, 1948

2:00 P.M. EXECUTIVE SESSION

3:00 P.M. SCIENTIFIC SESSION

Address of the President—Alton Ochsner. Factors Influencing Survival Rate in Primary Pulmonary Malignancy

20. **Decortication in Pulmonary Tuberculosis Including Studies of Respiratory Physiology**
Joseph Gordon and Edward S. Welles
21. **Decortication of the Unexpanded Tuberculous Lung Following Pneumothorax**
Joseph A. Weinberg and (by invitation) J. Dwight Davis
22. **Physiological Observations Concerning Decortication of the Lung**
George W. Wright, Lester B. Yee, Giles F. Filley, and Allan Stranahan (by invitation)
23. **Allergy of Tuberculosis, With Special Reference to Autotuberculinization**
A. R. Judd

Wednesday Morning, June 2, 1948

9:00 A.M. SCIENTIFIC SESSION

24. **Chronic Atelectasis and Pneumonitis of the Middle Lobe**
Donald L. Paulson and Robert R. Shaw
25. **Surgical Management of Pulmonary Coccidioidomycosis in Focalized Lesions**
Sam J. Greer (by invitation), James H. Forsee, and (by invitation) Hugh W. Mahon
26. **Changes in Blood Volume and Available Fluid Volume During Thoracic Operations**
Bernard J. Miller (by invitation), John H. Gibbon, Jr., and Frank F. Allbritten, Jr.
27. **Monaldi Suction in the Treatment of Bullous Emphysema and Large Pulmonary Cysts**
Jerome R. Head and (by invitation) Edward E. Avery
28. **A Review of Seven Years' Experience With Intracavitary (Monaldi) Drainage of Tuberculous Cavities**
Warriner Woodruff, Winfield O. Kelley, and (by invitation) Allan Stranahan

Wednesday Afternoon, June 2, 1948

2:00 P.M. SCIENTIFIC SESSION

29. **Coarctation of the Aorta**
    H. Brodie Stephens and (by invitation) Orville F. Grimes
30. **The Correction of Aortic Insufficiency in Dogs With an Artificial Aortic Valve**
    J. Moore Campbell (by invitation)
31. **Experiences With the Anastomosis of the Left Subclavian Artery to Left Pulmonary Artery for Pulmonary Stenosis**
    Emile Holman
32. **Development of Anastomosis Between Coronary Vessels and Transplanted Internal Mammary Artery**
    Arthur M. Vineberg
33. **An Experimental Study in Bronchial Anastomosis**
    Truxton Jackson, Phillip Lefkin (by invitation), William M. Tuttle, and (by invitation) Foster Hampton
34. **Mesothelial Mediastinal Cysts**
    Everett C. Drash

In addition to the papers listed here, two others have been submitted for the Rose Lampert Graff prize as follows:

Decortication of Nonexpandible Postpneumothorax Tuberculous Lung
Daniel A. Mulvihill and (by invitation) Robert Klopstock

Bilobectomy: Surgical and Anatomical Considerations in Resection of Right Middle and Lower Lobes Through the Intermediate Bronchus
Gustaf E. Lindskog and (by invitation) Liebow and Hales

Papers to Be Read by Title

1. **The Use of Streptomycin in the Preparation and Protection of Patients With Pulmonary Tuberculosis Treated by Extrapleural Thoracoplasty**
   James H. Forsee, and (by invitation) A. J. Neerken and Carl W. Tempel
2. **Phrenic Nerve Paralysis—Special Consideration of the Accessory Phrenic Nerve**
   Winfield O. Kelley
3. **Experiences With the Surgical Resection of the Human Carina, Tracheal Wall and Contralateral Bronchial Wall, in Cases of Right Total Pneumonectomy**
   Osler A. Abbott
4. **An Exact Method of Delineation of Bronchial Pulmonary Segments During Pulmonary Resection by Endobronchial Injection of Dyes**
   Charles P. Bailey
5. **Diaphragmatic Hernia**
   Herbert Willy Meyer
6. **Evaluation of Plombage in the Treatment of Chronic Pulmonary Disease**
   H. Ryerson Decker
7. **Emergency Management of Massive Pulmonary Hemorrhage**
   Alfred Goldman
8. **The Present Status of Bronchoscopy in Relation to Thoracic Surgery**
   Chevalier L. Jackson
9. **Lipoid Pneumonia Resulting From a Dermoid Cyst**
   A. Lincoln Brown
10. **Endobronchial Balloon for the Control of Bronchial Secretions During Lobectomy and Pneumonectomy**
    James D. Moody and George W. Newton (by invitation)
11. **Acute Putrid Pulmonary Abscess—the Later Results of Operative Treatment**
    Arthur S. W. Touroff and (by invitation) Robert A. Nabatoff, and Harold Neuhof
12. **Total Pleurectomy and Pneumonectomy for Cavitary Pulmonary Tuberculosis Complicated by Tuberculous Empyema**
    Daniel A. Mulvihill and (by invitation) Ralph Friedlander
13. **A Discussion of a Series of Operations for Coarctation of the Aorta and the Problems Involved**
    John C. Jones
14. **Transthoracic, Thoracoabdominal Portacaval Anastomosis**
    Victor P. Satinsky, H. P. Redondo Ramirez, and Lawrence Gilbert (by invitation)
15. **Intercostal Approach in Transpleural Operations**
    Joseph A. Weinberg and (by invitation) Alvin R. Kraus